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ANÆSTHESIA AND PULMONARY ATELECTASIS.[1]

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IT is very probable that an absorption atelectasis is the essential basis of most post-operative pulmonary complications, the various inflammatory, suppurative or necrotic changes being superimposed on it.[1][2][3] Localized and "patchy" atelectasis and spontaneous massive pulmonary collapse differ only in the extent of their distribution, the immediate cause being identical in all cases, even if numerous and variable contributory factors are involved. This primary factor is a localized or widespread stasis of the gaseous pulmonary contents, which is followed by their diffusion into the blood stream and collapse of the affected area. The speed and completeness of this collapse depend largely on the composition of the enclosed gases and the duration of their stasis. Ordinarily, air, with its high content of chemically inert nitrogen, provides an important, if not essential, safeguard against pulmonary collapse. Nitrogen, in comparison with oxygen, carbon dioxide and the gaseous and volatile anæsthetics, is absorbed very slowly from the pulmonary alveoli, the complete process taking from twelve to sixteen hours, whereas with the others it is a matter of minutes only.[4][5][6] Hence artificial atmospheres containing a great excess of oxygen will undergo very rapid absorption in appropriate circumstances.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on June 29, 1939.

TABLE I.

Average Absorption Times of Gases and Vapours from Lung Inflated to Full Inspiration. (After Coryllos and Birnbaum.[4])

Agent.	Time.
Ethyl ether	1- 3 minutes
Carbon dioxide	4 minutes
Oxygen	15 minutes
Ethyl chloride	10-17 minutes
Ethylene	13-29 minutes
Nitrous oxide	17-35 minutes
Nitrogen	16 hours
Hydrogen	18 hours
Helium	+ 26 hours

Contributory Factors.

Respiratory Obstruction.

Obstruction at some point in the respiratory tract is the commonest cause of gaseous stasis in the lungs, and its site determines the location and extent of the consequent alveolar collapse.[3] The usual site is in one or more of the bronchi or bronchioles, but may be at a higher level. There are various causes of obstruction. The chief of these causes are: inhalation of foreign material from the pharynx (secretions, vomitus, blood and solid objects); hypersecretion in the tracheo-bronchial tree; inundation with blood, pus, necrotic or hydatid material during the course of lung operations; the insertion of bronchial occlusive appliances in such operations as pulmonary lobectomy;[7] laryngeal and possibly bronchial spasm;[8][9][10] and displacement of the tongue. In addition, various forms of partial obstruction are often significant in that they impair pulmonary ventilation and favour the operation of other factors. This group comprises such conditions as nasal congestion, spasm of the jaw muscles, inadequate reflux around endotracheal catheters when the air inflow fails to meet tidal requirements, kinking or compression of wide-bore (Magill) endotracheal tubes, the presence of tumours, œdema, surgical emphysema *et cetera*. Their efficient relief is frequently neglected in practice, and this denotes an imperfect appreciation of the possibilities they involve. In every respect provision of a clear airway remains a basic principle of all forms of anæsthesia, both general and local.

Magill's method of tracheal intubation is a valuable safeguard against the inhalation of foreign material, especially during operations in the mouth, nose and throat. It is described as endotracheal inhalation to distinguish it from the more commonly used insufflation technique. As large a tube as the glottis will accommodate should be passed, and it is usual to insert a pharyngeal pack of moist gauze or small marine sponges to ensure a good seal-off. The method is of special value when inundation with vomitus is likely to occur during the operation or when the mouth and pharynx will not be accessible for clearance by suction. The hazards of dental surgery are greatly reduced by its employment.[11]

Most inhalational anæsthetics, with the possible exceptions of nitrous oxide and chloroform, cause a hypersecretion in the respiratory tract. This occurs in aggravated form with ethyl ether because of its irritant properties, and sometimes of itself constitutes an acute emergency. Ethyl chloride, vinyl ether and cyclopropane tend to cause a moderate amount of hypersecretion, but they are irritant only in very high concentrations. Laryngeal and perhaps bronchial spasm are also possibilities when irritating agents are employed in excessive concentrations.

Control of hypersecretion, and perhaps of spasm,[12] is readily effected by the preliminary administration of atropine sulphate or hyoscine hydrobromide. The dosages employed should be sufficient only to inhibit excessive secretion and not so large as to interfere with the normal mechanism of pulmonary drainage. In general it is rarely necessary to exceed 0·43 milligramme ($^1/_{150}$ grain), provided that the solution used is freshly prepared. The use of atropine has been questioned on the grounds that it causes the patient some discomfort, increases the metabolic rate and oxygen demand, disturbs the balance of the autonomic system[13] and favours the secretion of extremely tenacious mucus as its effect passes off.[3] On the whole, however, the advantages obtained from its administration preponderate, provided that abuse is avoided. It certainly should not be given post-operatively in repeated doses in an effort to control persistent hypersecretion, being likely to aggravate the dangers involved. The viscosity of secretions is also said to be dependent on the virulence of infecting organisms (pneumococci) that may be present.[3]

Impaired Pulmonary Drainage.

The delicately adjusted mechanism of pulmonary drainage is very easily disturbed by the circumstances of anæsthesia and operation. Respiratory depression, prolonged immobility, diminished ciliary activity, impaired efficiency of coughing, pain *et cetera*, all tend to the retention of abnormal secretions. The recumbent posture, commonly adopted during recovery from the effects of anæsthesia, is an important accessory factor. It is remarkable how rarely the natural posture for sleep, the lateral decubitus, is employed in connexion with unconscious patients. The lateral posture not only ensures a clear airway, but changes from side to side effect a redistribution of the thoracic and abdominal contents, while drainage from the uppermost lung is facilitated. There are few surgical contraindications to its employment, and a wider appreciation of its value is desirable. It must be emphasized that a complete lateral position is recommended, and not merely a twisting of the head to one side, a proceeding which frequently aggravates the condition it is intended to relieve. By virtue of its broncho-dilator action, ephedrine may be of value in favouring pulmonary drainage after operations, and the administration of expectorants, such as ammonium carbonate and potassium iodide, may be indicated.

Condition of Patient.

A careful preliminary assessment of the patient's general condition is of the utmost importance, so that anything known to favour the development of atelectasis shall not be overlooked. States of debility, wasting, cachexia and general weakness are associated with an increased incidence of the condition.[14] Infections of the upper part of the respiratory tract, oral sepsis, bronchitis,[10][21] thyreotoxicosis[15] and lung diseases in general are further predisposing factors. Reduced vital capacity from abdominal effusions and tumours and from muscular rigidity due to intraabdominal inflammatory conditions are also significant. When patients are classified under risk headings, distinguished as "A", "B", "C" or "D" as the estimated tolerance to anæsthesia and operation varies, a greater incidence of complications is found to occur in the poor-risk cases.[16][21]

In severe cases of thyreotoxicosis patients often exhibit hypersecretion of tenacious mucus quite apart from anæsthesia or operation; this is a feature of the so-called thyreotoxic crisis.[17] It is perhaps extremely susceptible to activation by the various anæsthetic agents, passage of endotracheal tubes and surgical manipulations about the trachea. Should it develop during or after operation, an extremely grave situation becomes established, especially since coughing will be greatly impaired by pain. It is customary not to pass endotracheal tubes in such cases so as to avoid at least one of the possible causative factors. Whether atropine should be used as a preliminary step or not is a very open question. Recent suggestions that cyclopropane particularly aggravates the condition are not well substantiated, especially since it is not of invariable occurrence and its maximum development is usually seen several hours after operation, when most, if not all, of the anæsthetic has been eliminated. A brief account of two recent cases of pulmonary atelectasis following thyreoidectomy under cyclopropane anæsthesia follows.

CASE I.—A female patient, aged fifty-six years, suffered from moderately severe thyreotoxicosis of seven months' duration. Her condition was much improved by three and a half weeks' rest, bromides and Lugol's iodine. The average pulse rate decreased from 100 to 80-84 per minute. The basal metabolic rate fell from +39% to +36%. A blood count revealed 4·53 million erythrocytes per cubic millimetre; the hæmoglobin value was 63% (8·9 grammes *per centum*), indicating impaired oxygen-carrying capacity. She would evidently constitute a fair "surgical risk". No formal assessment was made, but classification under the heading of "C" risk was warranted. Subtotal thyreoidectomy was performed under nitrous oxide-oxygen and cyclopropane anæsthesia. Premedication consisted of morphine sulphate, 11·0 milligrammes (one-sixth of a grain), given about one hour before operation. No atropine was given. Anæsthesia was uneventful. The operation record states that air seemed to be sucking in and out of the dome of the left pleura towards the end, but no hole was found. A moderate reaction occurred on the evening of operation, the pulse rising to 100 per minute. From the next day and until death six days later there was increasing fever with tachycardia and dyspnœa. A small area of emphysema was noted in the left anterior triangle of the neck. Pain over the lower ribs led to the application of strapping. Râles and rhonchi were noted at the bases of both lungs. Three days after operation there was a troublesome cough with yellow sputum. The patient was considered to have a very early lobar pneumonia. Despite continuous oxygen therapy, chemotherapy, the administration of Lugol's iodine and, on the last day, adrenaline, cyanosis increased and the patient died.

At autopsy twenty-two hours after death the lower lobe of the right lung was found partly and the lower lobe of the left lung entirely collapsed. An acute tracheo-bronchitis was present. The lower lobe bronchi on both sides were completely filled with mucinous, semi-fluid, non-purulent exudate. No emboli were found. Pneumococcus Type XV (mild virulence) and Gram-positive and Gram-negative bacilli were grown on culture. Mild toxic degenerative changes were noted in the liver and kidneys.[1]

CASE II.—A female patient, aged eighteen years, suffered from rather severe thyreotoxicosis of eighteen months' duration. She had a history of previous asthma. Her condition was much improved by five and a half weeks' rest, sedatives and Lugol's iodine. The average pulse rate decreased from 110 to 80-84 per minute. The basal metabolic rate was +37%. A blood count revealed 4·9 million erythrocytes per cubic millimetre; the hæmoglobin value was 69% (9·8 grammes *per centum*), indicating impaired oxygen-carrying capacity. An electrocardiogram suggested a myocardial defect. The systolic blood pressure was 130 and the diastolic pressure 80 millimetres of mercury. The patient was very alert and "fidgety", exhibiting tachycardia on slight provocation. Intravenous administration of calcium gluconate was performed four and two days before operation with a view to lessening bleeding. Tolerance to anæsthesia was assessed as being "fair", which, in conjunction with an operation of long duration, led to her classification as a "C" risk. Premedication consisted of "Avertin", 150 milligrammes per kilogram (7·5 grammes), given one and a quarter hours before operation in 3% solution, and atropine, 0·65 milligramme (one one-hundredth of a grain), given one hour before operation. The lateral posture was maintained throughout the induction of basal narcosis and during transportation. Subtotal thyreoidectomy was performed under nitrous oxide, oxygen and cyclopropane anæsthesia, given by the closed circuit carbon dioxide absorption method, occupying two hours. Some stridor and a good deal of stertor complicated the early part of the procedure, and helium was added to the gaseous mixture with beneficial results, making positive pressure and inflation very much more effective. At all times hyperoxygenation was prevented by the use of nitrous oxide and helium as diluents, but no suboxygenation was allowed to occur. The accompanying chart demonstrates the really excellent condition of the patient throughout the operation, and especially at the end. She was semi-conscious and somewhat restless within fifteen minutes. On the evening of operation hypersecretion of tenacious mucus began to occur, and next day the classical picture of a massive left lobar collapse had developed—the left side of the chest was fixed, the breath sounds were weak and the heart was displaced to the left. There were also dyspnœa and some cyanosis. The following day a large hæmatoma had formed in the left side of the neck. Meanwhile, the lung condition had begun to improve, and within a few days its spontaneous resolution had occurred. The patient was discharged from hospital, well, seventeen days after operation.[2]

Neither of these cases provides incontrovertible evidence to incriminate cyclopropane. They merely demonstrate the necessity for extreme care in all respects when this grave condition of thyreotoxicosis is being dealt with. The use of voluminous restrictive dressings and the neglect of posturing likely to favour pulmonary drainage are of significance.

[1] Acknowledgement is made to Dr. George Bell and Dr. Hugh Hunter for permission to report this case.

[2] Acknowledgement is made to Dr. W. E. Fisher and Dr. C. E. Winston for permission to report this case.

The rigid semi-recumbency enforced after thyreoidectomy is a potent aggravating factor. Price-Thomas reports a typical case as occurring after thyreoidectomy performed under local analgesia and paraldehyde-morphine narcosis.[10]

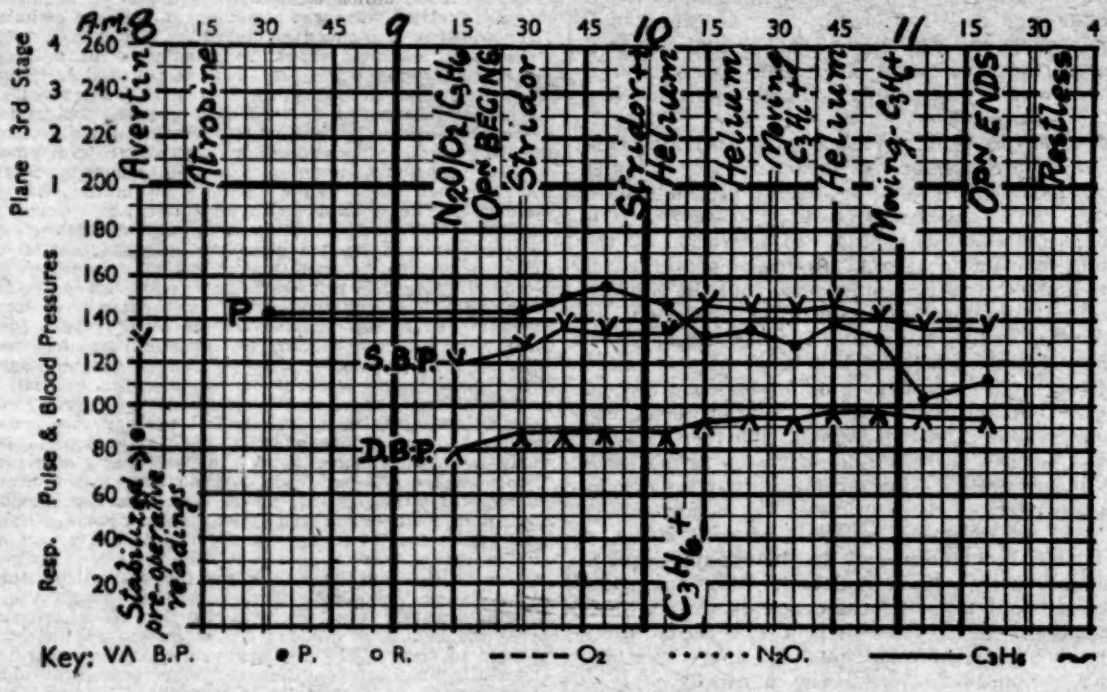
Premedication and Basal Narcosis.

Excessive premedication and the abuse of basal narcosis, involving prolonged quiescence, immobility and respiratory depression, especially after operation, are extremely significant factors.[18] The use of "Avertin" in connexion with operations in the upper part of the abdomen has frequently had disastrous consequences. This drug has a limited utility (young children, thyreotoxicosis, neurosurgery) and in general full basal narcosis is best procured with a short-acting barbiturate, such as "Pentothal Sodium".[19] Partial respiratory obstruction from faulty posturing, displacement of the tongue *et cetera* often complicates the situation. Here again the lateral decubitus is invaluable. If necessary, analeptics ("Coramine", "Picrotoxin" or "Cardiazol") may be given intravenously in large dosages for their awakening effect and stimulation of the respiratory and vasomotor nervous centres.[20] The increased incidence of complications after major operations performed under local analgesia is probably associated with the necessity for heavy premedication in such circumstances.

Surgical Requirements.

Excessive depth and duration[16][21][22] of anæsthesia and the employment of certain unfavourable postures during operations favour the occurrence of gaseous stasis and alveolar collapse. The use of unnecessarily high lateral or posterior supports during renal and biliary operations is deprecated in this connexion. The same criticism applies to the steep Trendelenburg position and firm abdominal packing sometimes employed in gynæcological surgery.

The deeper levels of general anæsthesia are characterized by a progressive ascending paralysis of the intercostal muscles.[22] The addition to this of diaphragmatic embarrassment gravely compromises the situation. The prolonged maintenance of absolute relaxation during general anæsthesia



involves delayed restoration of muscular tone, which includes in its effects the muscles of respiration and may persist for several days after operation.[3] The vital capacity of the lungs may thus be reduced by as much as 75% of the normal after some operations.[24] Discrimination in the use of powerful agents like ether and chloroform is therefore an obvious necessity, and their utility as adjuvants to the less potent gaseous anæsthetics deserves consideration. The surgeon must be prepared at times to forgo the advantages of full muscular relaxation or at most to employ them for the briefest possible periods only.[22] The flexibility of cyclopropane is of great value in this connexion, making possible rapid variations in depth as may be required.

Site of Operation.

The site of operation is an extremely significant factor. The incidence of atelectasis and pulmonary complications in general is highest after operations

in the upper part of the abdomen.[1] This is largely due to a reflex and voluntary inhibition of diaphragmatic activity, while the efficiency of coughing is greatly impaired because of pain. The almost doubled incidence in males is probably determined by their dependence on the diaphragmatic element in respiration. The application of tight binders to fix dressings is a further embarrassment to proper lung expansion.

Similar considerations must also apply in modern thoracic surgery, which may involve in exaggerated degree the problems of impaired pulmonary ventilation, inundation and gaseous stasis with rapid absorbability because of the high oxygen concentrations demanded by the restricted tidal exchanges. Preliminary bronchoscopic aspiration, tracheal intubation with a wide-bore Magill tube, employment of a closed circuit carbon dioxide absorption method which permits of rapid lung inflation and the maintenance of positive intrapulmonary pressures, and provision of facilities for immediate deep suction are some of the essential precautions. It is here necessary to stress the danger of the application of suction direct to Magill tubes so inserted. Serious deflation of the lungs will result, especially if ingress of air around the tube is prevented or impeded by a pharyngeal pack. The passage of a small catheter down the lumen of the tube is a safe and usually efficient procedure for the removal of foreign material. At times, however, nothing less than the use of a bronchoscope and the application of powerful suction will overcome the acute emergency which gross inundation constitutes.

Anæsthesia.

Although the anæsthetic is often unjustly blamed for complications which develop during and after surgical operations, it cannot be entirely absolved of considerable direct or indirect responsibility. The term "post-anæsthetic pulmonary complications", however, is frequently a misnomer in all respects, except as regards time.[25] Evidently the association of the inhalational route of administration with subsequent lung changes encourages the too obvious explanation. Lung complications have an increased incidence when local and spinal analgesia[15][16][21] and basal narcosis[17] are employed as indiscriminately as inhalational anæsthesia commonly is.

Apart from their absorbability,[4] the various inhalational anæsthetic agents are of little direct significance in the occurrence of atelectasis. Indirectly they may originate or participate in the establishment of a variety of conditions which are of great ætiological importance. The potency and irritant properties of ethyl ether favour the operation of a wider range of predisposing factors than is usual with the various anæsthetic gases. The great affinity of fatty tissues for cyclopropane favours its rapid absorption from the pulmonary alveoli, which may be significant if the proportion of inert gas in the inhaled mixture is low.

The following case report demonstrates the occurrence of atelectasis after ether anæsthesia administered in accordance with standard practice.

CASE III.—A female patient, aged seventy-five years, suffered from hypertension, impending congestive cardiac failure and cholecystitis. She had had a "stroke" eight years previously. Some recent loss of weight had occurred. She voided sputum periodically. Examination disclosed precordial hyperæsthesia, an enlarged heart, an accentuated aortic second sound, and a systolic blood pressure of 200 and a diastolic pressure of 140 millimetres of mercury. Some œdema of dependent parts was present. Rhonchi and crepitations were present at both lung bases. X ray examination revealed chronic bronchitic changes. A trace of albumin was found in the urine. A blood count revealed 3·93 million erythrocytes per cubic millimetre; the hæmoglobin value was 63% (8·9 grammes *per centum*), indicating impaired oxygen-carrying capacity. Temperature and pulse and respiratory rates were normal. No formal assessment of risk was made; tolerance was obviously poor, and classification as "D" risk inevitable. No premedication was used; no atropine was given. Cholecystectomy was performed under endotracheal ether anæsthesia (insufflational). Much difficulty was experienced because of hypersecretion and vomiting during maintenance. The post-operative period until death six days later was characterized by gross hypersecretion, irregular pyrexia and rising pulse and respiratory rates. Dyspnœa was present on the second day, and dulness and weak breath sounds were noted at the base of the right lung. Evidently, with the object of controlling secretions, four hypodermic injections of atropine sulphate, of dosages ranging from 0·43 to 0·86 milligramme ($^1/_{150}$ to $^1/_{75}$ grain), were given during the five days preceding death.

Autopsy twenty-two hours *post mortem* disclosed elevation of the right cupola of the diaphragm to the level of the fifth rib. There was a small right pleural effusion (half a pint). The right middle and lower lobes were entirely collapsed and there was a localized area of collapse in the left lower lobe, near its base. Firm compression caused expulsion of thick gelatinous plugs from the larger bronchi in the affected areas. The heart showed undue fatty deposition, the coronary vessels were sclerosed and the myocardium was flabby. The aorta was calcareous.[1]

Morison[8] reports six cases following ether anæsthesia, in each of which atropine sulphate, 0·65 milligramme (one one-hundredth of a grain) had been given beforehand.

The belief that local or spinal analgesia[10] is invariably the best method available for poor-risk cases is fallacious. Excessive segmental ascent of spinal analgesia, especially when long-acting agents like "Percaine" are used, introduces several predispositions to atelectasis. Paresis of the intercostal muscles may be prolonged and vital capacity much reduced; the necessity for subsequent recumbency impairs pulmonary ventilation and drainage; atropine, used to offset the tendency to vagal hyperactivity, favours viscidity of secretions; and the site of operation may cause inhibition of the diaphragm and of coughing. Here again few valid reasons exist against the use of alternating lateral postures during the recovery period. The following case notes are illuminating.

CASE IV.—A male patient, aged sixty-seven years, had suffered for two years from partial urinary obstruction from an enlarged prostate gland. A moderate degree of debility was present; the patient was very thin. The

[1] Acknowledgement is made to Dr. W. E. Fisher and Dr. C. E. Winston for permission to report this case.

blood urea content was 56 milligrammes *per centum*. His condition was much improved after a period of continuous bladder drainage. He was a heavy smoker, with chronic bronchitis and copious sputum. X ray examination of his chest revealed no abnormality. A rather hurried assessment was made and classification as a "B" risk was adopted. Subsequent review indicates that "C" classification would have been more appropriate. Premedication was carried out with morphine sulphate, 11 milligrammes (one-sixth of a grain), and atropine sulphate, 0·65 milligramme ($^1/_{100}$ grain), given one and a half hours before operation. On his arrival in the theatre, twenty minutes before operation, ephedrine sulphate, one cubic centimetre of a 3% solution (30 milligrammes), was given intramuscularly. Cystotomy and partial removal of a malignant prostate gland were performed under spinal analgesia, heavy "Percaine" solution, 1 in 200, two cubic centimetres (10 milligrammes), being used. Puncture was effected in the third lumbar interspace with the patient sitting. The epidural aspiration sign was positive. The solution was injected without barbotage, and the patient was postured on a sandbag and pillows, with the thighs flexed on the abdomen for "medium rise", that is, to the level of about the tenth thoracic segment. The ultimate upper limit of analgesia was at the sixth thoracic segment. Operation was uneventful, the duration being one hour and ten minutes. The pulse rate fell from 100 to about 74 per minute during its course. To avert headache the patient was kept recumbent for twelve hours after operation. The next day his temperature was raised, and on the second day it reached 39·4° C. (103° F.). Drainage from the bladder was clear. The movements of the right side of the chest were limited, but no gross auscultatory signs were present. On the third post-operative day the sputum, which had been tenacious, became looser. Profuse expectoration occurred and an improvement in his condition was almost immediate. Apart from mild rises of temperature to about 37·2° C. (99° F.) on the following three evenings, the subsequent course was uneventful.[1]

Evidently the following factors were involved: debility, heavy smoking, chronic bronchitis, the use of atropine in large dosage, excessive ascent of analgesia, the use of a long-acting anæsthetic drug, and prolonged post-operative recumbency.

All these cases have been noted within the past six months, and two of them (II and IV) were consecutive in my series, although in different hospitals. This suggests a frequency of the condition not commonly realized.

Hyperoxygenation.

During recent years, and especially since the introduction of the carbon dioxide absorption technique, an increasing tendency to employ oxygen as a vehicle for inhalational anæsthetic agents has developed. Superficially it appeared immensely advantageous to be able to give an agent such as ethyl ether or cyclopropane with oxygen concentrations rising as high as 80% or 85%, and in some circumstances it is indeed so provided that the concurrent dangers are recognized and guarded against. Ordinarily, however, it is most undesirable to dispense with the normal quota of inert nitrogen in respired atmospheres, since this, by virtue of its slow absorbability,[4] greatly delays the progress of alveolar collapse when gaseous stasis occurs in any part of the lungs.[6] Oxygen-rich atmospheres will undergo rapid absorption in such circumstances.

[1] Acknowledgement is made to Dr. A. C. Telfer for permission to report this case.

Recently several cases of massive pulmonary collapse have been reported as occurring during or after anæsthesia with cyclopropane,[22][26] and an impression has gained ground that the gas itself was responsible. The onset has at times been of appalling rapidity, resulting in an anoxæmic emergency of the first order. At other times moderate hypersecretion has occurred and a typical localized atelectasis has developed subsequently. The employment of mixtures of oxygen and cyclopropane only in proportions suitable for the induction of anæsthesia carries an extremely grave risk should stasis occur from obstruction or any other cause. Exactly the same danger exists with similar mixtures of oxygen and ethyl ether, chloroform or vinyl ether. As has been pointed out in recent correspondence[27][28][29] air, or an oxygen mixture resembling it very closely in inert gas content, is the ideal vehicle for gaseous or volatile anæsthetics.

An interesting recent therapeutic development has been the use of helium in conditions of respiratory embarrassment, especially in *status asthmaticus*.[30][31][32][33] Mixed with oxygen in the same proportion as that of nitrogen in air it greatly reduces the density and the inertia of respired atmospheres, so facilitating their passage through constricted orifices. The principle is of value in anæsthesia,[34][35] especially when some irremediable partial obstruction or embarrassment to respiration exists. Helium is quite inert in the body and its absorption time is slow.[4] Its use in anæsthesia lessens the amount of energy expended by the patient in breathing, effects a much more rapid and complete diffusion of oxygen while preserving the safeguard of an adequate inert gas content, and greatly facilitates emergency lung inflation and artificial respiration. It is of great value when tidal exchanges are gravely restricted, for example in thoracic surgery. In all such special circumstances it would constitute a valuable safeguard against pulmonary collapse, while its diffusibility and lightness would greatly facilitate gaseous exchanges.

The situation with nitrous oxide is different in that the exclusion of all nitrogen is essential so as to provide the greatest possible concentration of the gas and yet to minimize the suboxygenation inevitable in this form of anæsthesia. Tissue saturation with nitrous oxide, however, is rapidly effected; its affinity for fats is low, and once anæsthesia is established little danger of excessive absorption exists. Contrary to Jones and Burford's views,[26] therefore, I believe that nitrous oxide is a safe diluent when ethyl ether, chloroform, cyclopropane or vinyl ether is used as an adjuvant. Of course, no suboxygenation should be permitted, and no attempt should be made to "wash out" nitrogen at the beginning of the induction, although leakages and diffusion tend to have this effect as the administration proceeds.[26] There is no reason why air should not be used for such dilution purposes in closed circuit anæsthesia.

The custom of "flushing out" with oxygen or carbon dioxide and oxygen mixtures as a termination to anæsthesia is not without risk in view of the foregoing considerations. If the production of hyperpnœa is desirable, the employment of mixtures of carbon dioxide and air are far safer, and this should be borne in mind in the application of post-operative inhalational therapy.[5]

Conclusion.

The obvious query, "Why not stick to the good old open and insufflational methods?" ignores their numerous disadvantages and the fact that they often fall short of the requirements of modern surgery. It must be admitted that the increasing complication in methods of anæsthesia makes their application a matter for expert handling and not of casual dalliance; but the results obtained are worth the trouble. Simplicity of method is by no means an invariable preventive of atelectasis or other complications, often being rather an embarrassment because of the lack of facilities for dealing with emergencies.

The different factors involved in the production of atelectasis may operate in any variety of combination,[9] and sometimes it is impossible to take effective precautions against all of them. A careful preliminary assessment and preparation of the patient and a rational choice and efficient application of anæsthetic technique are essential safeguards. Beyond this the intelligent and reasonable cooperation of all concerned in the conduct of surgical operations is indispensable.

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MASSIVE COLLAPSE OF THE LUNG.[1]

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THE condition to be discussed has been described under various titles, such as acute massive collapse of the lung, acute lobar collapse, post-operative massive atelectasis, post-operative pulmonary atelectasis, obstructive massive atelectasis, and so on.

The title "massive collapse of the lung" was originally used by William Pasteur,[1] of Middlesex Hospital, in 1908, when he first gave a clear description of this condition as a clinical entity.

The term "atelectasis" signifies incomplete dilatation; and although strictly speaking it should be

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on une 29, 1939.

confined to a congenital condition in which the lung fails to pass from the fœtal to a fully expanded condition, in actual practice it has come to be synonymous with collapse or with the passage from a condition of complete to one of incomplete expansion.

In many cases of atelectasis the mechanism of its production is readily understood, as, for example, in cases of pressure on the lung from without by a pleural effusion or by a pneumothorax (compression atelectasis) or in cases of bronchial obstruction (obstructive atelectasis). Pasteur,[1] however, defined massive collapse of the lung as a condition of total deflation of a large area of lung tissue of sudden onset, in the absence of any signs of obstruction of the airway or of any known cause of compression.

An important point to be determined, then, will be whether acute massive collapse of the lung is a condition *sui generis* or whether it is merely a special manifestation of pulmonary atelectasis.

It must also be emphasized that in massive (atelectatic) collapse the lung does not leave the chest wall. In its collapsed or atelectatic state the lung occupies a smaller space than it did when fully expanded. To compensate for the space lost from collapse of the lung the chest wall is depressed, the trachea, heart and mediastinal structures are drawn over to the involved side and the diaphragm is pulled up. The loss of air content produces consolidation of the lung fully as dense as that of lobar pneumonia.

Incidence and Mechanism.

Gairdner gave a clear description of atelectasis about the year 1850, and in 1853 gave the following as the three causes of pulmonary collapse in infancy: mucus in the bronchi, weakness of the respiratory power and inability to cough and thus remove the mucus.

The effect of bronchial obstruction was first demonstrated experimentally by Mendelssohn in 1845, and later by Lichtheim in 1879.

It was not until Pasteur[1] published his series of articles (1908 to 1914) that considerable clinical interest was stimulated in the recognition of massive collapse of the lung. Pasteur's earlier observations were made on patients suffering from diphtheria. In a lecture delivered in 1908 he referred to a series of sixty-four cases of post-diphtheritic paralysis observed by himself, in twenty-eight of which the diaphragm was affected, with a fatal result in fifteen cases. An autopsy was performed in eight of these. In five there had been clear evidence of paralysis of the diaphragm for several days before death, and in each of these the lower lobe of the right lung was completely collapsed, sinking entirely in water, of a deep blue colour and absolutely devoid of air. Pasteur believed that the pulmonary condition found in these cases was due to a failure of respiratory power, namely, complete arrest of diaphragmatic action due to paralysis of the diaphragm.

Later Pasteur turned his attention to collapse of the lung following abdominal operations. On this subject he may be quoted *verbatim*:

> When I began to examine patients suffering from "post-operative pneumonia" I soon realized that the signs and symptoms they presented were not quite on all fours with those of lobar pneumonia as met with in medical wards. Although the physical signs may be indistinguishable the symptoms and course of the affection are frequently different. The fact that this "pneumonia" was so frequently situated at the right base led me to suspect that it might be massive collapse rather than pneumonic consolidation.

Pasteur believed that post-operative massive collapse was the result of (reflex) arrest of the action of one half of the diaphragm. It is interesting to read that prior to his description of this condition the surgical records of the Middlesex Hospital furnished no examples of massive collapse; but subsequently there was a gradual increase in the number of cases observed, with a corresponding diminution in those of post-operative pneumonia. There is much more in this than a mere change of name, providing the diagnosis of massive collapse is never made unless the apex beat is displaced towards the affected lung.

In the cases of massive atelectasis following diphtheria in which Pasteur recorded his *post mortem* findings, he was unable to find any evidence of bronchial obstruction which would account for the condition in any instance.

Pasteur's patient clinical investigations and his brilliant description of this condition will always be remembered, although his original contention that it was brought about by paralysis or reflex arrest of the diaphragm is not now generally accepted. On this point it is interesting to note that true massive collapse of the lung seldom, if ever, follows section of a phrenic nerve carried out as a therapeutic measure in patients suffering from pulmonary tuberculosis.

In 1914, soon after Pasteur's last paper, Elliott and Dingley[2] described eleven cases of post-operative collapse which they stated agreed closely with Pasteur's description. They stressed the occurrence of mucopurulent sputum and fever, and finally came to the conclusion that the massive collapse in their cases was a direct result of occlusion of the bronchi by bronchial secretions, the circulating blood absorbing the imprisoned alveolar air distal to the obstruction. Elliott and Dingley were therefore the first of the modern writers in favouring the theory of bronchial obstruction.

About 1919 and 1920 Rose Bradford[3] described a large series of cases of massive collapse of the lung following non-penetrating wounds of the chest seen during the World War. Wounds of the abdomen, pelvis, buttocks and legs were followed by this complication even when there was no lesion of the abdominal or pelvic cavities. In wounds of the chest the outstanding fact was that massive collapse in its most extreme form, involving the

whole of one lung, was observed following small wounds, limited absolutely to the chest wall. Further, the collapse occurred not infrequently on the side of the chest opposite to that injured, even when the wound was of this trivial character and the collapse involved the entire lung. In such cases Rose Bradford found no evidence indicating injury of the lung itself, and no evidence of any gross obstruction to the larger bronchi. He pointed out that it was very difficult to see how a unilateral injury limited to the chest wall, in a healthy man, could within twenty-four hours produce an obstruction of a main bronchus of the opposite lung sufficient to cause collapse of the lung. He also stated that there was definite *post mortem* evidence that massive collapse might be present when no obstruction of the main bronchi was found; and he concluded that bronchial obstruction could not be regarded as the cause of massive collapse of the lungs seen after injuries to the chest wall. In his opinion the collapse of the lung was more readily explained as a result of the immobility of the chest wall produced in some obscure reflex manner by the injury to the neighbouring parts, or that, while it seemed incredible, a condition of reflex spasm of the bronchi might be set up which caused bronchial obstruction and then massive collapse.

In 1925 Jackson and Lee,[4] writing on acute massive collapse of the lung, discussed its mechanism and its relation to foreign bodies in the bronchi and post-operative complications. It is impossible to read their papers without realizing the supreme importance of bronchial obstruction. Jackson emphasized that the symptoms and the radiological appearance of massive collapse were duplicated in cases of foreign body in the bronchi; indeed, the radiological appearance of collapse may be one of the diagnostic features in favour of the presence of a non-opaque foreign body in the bronchus. Early removal of the foreign body may be followed by rapid disappearance of the symptoms and of the shadow seen in the skiagram. Similarly, Jackson held that in pneumonia following tracheotomy there was no true pneumonia, and stated that he had seen the signs of "pneumonia" disappear in a few minutes after the removal of obstructing secretions from the bronchi.

In 1925 he had found *post mortem*, in two cases of post-operative massive collapse, collections of mucus in the bronchi such as to cause complete obstruction. He also reported that he had seen such obstructing masses *in vivo* and that such mucous masses or plugs had been removed through a bronchoscope, and it had been proved that after such removal the lung returned to its normal condition.

It was Jackson who first suggested that bronchoscopic examination might not only determine the ætiology of this condition, but also serve as a therapeutic measure. At the same time Jackson and Lee recognized that another factor common to conditions in which acute massive collapse of the lung was associated, was partial or complete arrest of the respiratory movements. Thus in pleurisy, pneumonia, diphtheritic paralysis of the diaphragm, or the voluntary or reflex inhibition of the diaphragm and abdominal muscles after upper abdominal operations, this mechanism is obvious. Just as important, and more constant, is embarrassment of respiratory movements resulting from unnatural postures and prolonged rest after abdominal operations and from accidental injuries. Decreased aeration may result in an increase in the bulk of bronchial secretions. If these secretions increase and the cough reflex fails to expel the mucus, the lumen of a large bronchus may become completely obstructed. Such mucus obstruction will act in exactly the same way as a foreign body. Jackson described the cough reflex as "the watchdog of the lungs", and he emphasized the effect of posture, of severe post-operative pain and of the generous preoperative and post-operative use of morphine upon the expulsive cough necessary to clear the bronchial tree of secretion.

As mentioned before, the experiences and writing of Chevalier Jackson emphasize the importance of bronchial obstruction in the production of massive collapse of the lung. But, although bronchial obstruction admittedly explains nine-tenths or more of these cases, there are some which are difficult of acceptance on these grounds, and it cannot be said that in every case of acute massive collapse the bronchi will be found to be obstructed, although in any autopsy on subjects dying from this condition the bronchial tree should be thoroughly examined.

In 1927 Bergamini and Shephard,[5] of the Bellevue Hospital, New York, reported two cases of sudden death during operation, with autopsy findings of bilateral massive collapse of the lung. This is the first record in the literature to suggest that massive collapse, which is usually a benign unilateral condition occurring from a few hours to a few days after operation, may occasionally be bilateral, and may occur at or immediately after operation as an acute tragic complication causing sudden death. These authors pointed out that the extreme rapidity of onset in such cases tended to disprove the obstructive theory of the ætiology. *Post mortem* examination in their two cases revealed no evidence of gross obstruction to the bronchi of the collapsed lungs. The fact that their observations were confirmed by *post mortem* examination is important, because the pathological data obtained on the subject of massive collapse are very meagre.

About 1927, Sante,[6] of St. Louis, described cases of massive collapse of the lung. In his papers he wrote with particular reference to treatment of this condition. He held that treatment was simple, and consisted in the rolling of the patient backwards and forwards on the uninvolved side. He stated that no other treatment was necessary and that this simple procedure had proved to be successful in all instances in which he had used it in promptly reestablishing aeration of the lung. He

emphasized that the procedure was simple and available to all, and free from the risk entailed by bronchoscopic examination as recommended by Jackson. He also suggested the prophylactic value in post-operative treatment of the changing of the patient's position from one side to the other repeatedly during the first few days after operation.

In 1928, Lee, Tucker and Clerf,[7] of Philadelphia, writing on post-operative massive atelectasis, stated that they believed that 70% of cases of so-called post-operative and post-anæsthetic pneumonia were varying degrees of atelectasis. After studying 33 cases of post-operative massive atelectasis, they formed the opinion that two factors had been constant: first, a thick, viscid bronchial secretion, and, secondly, some inhibition of coughing. They believed that because of the thick tenacious character of the bronchial secretion and the inability or the disinclination of the patient to clear it from the bronchi, it accumulated in the dependent portions of the bronchial tree until at some point the lumen was completely occluded.

Clinically they stated that they had demonstrated that if this obstruction could be overcome, by making the patient cough, by a change in position as suggested by Sante, by vigorous shaking, and in the case of young children by actual spanking, and an airway established past the obstruction, the patient might, temporarily at least, free the bronchial tree of large masses of secretion and thus reinflate the pulmonary tissues. In eight cases they found it necessary deliberately to aspirate through a bronchoscope the obstructing portion of this bronchial secretion, and in each case immediate aeration and reinflation of the lung followed. The same authors experimentally produced atelectasis in a dog by introducing into its main bronchus a specimen of bronchial secretion removed by bronchoscopic drainage from a case of post-operative massive atelectasis.

Reference must be made to the work of Coryllos and Birnbaum,[8] who produced experimental bronchial occlusion in dogs by means of small balloons, which were inserted into the bronchi and then inflated. They demonstrated the fact that when bronchial occlusion was produced experimentally some hours elapsed before massive collapse occurred. During this period the air imprisoned distal to the obstruction was being absorbed by the lung, provided the circulation in the lung was intact and the alveolar epithelium undamaged. They also showed experimentally that a great variation existed in the rate of this absorption. Certain anæsthetic gases tested were absorbed in a few minutes, as were oxygen and carbon dioxide. Inert gases, such as nitrogen, hydrogen and helium, required from eighteen to twenty-six hours for absorption. Their experimental and clinical work led them to believe that the only cause of massive collapse was complete occlusion of a bronchus by a plug of mucus acting as a foreign body. In 1930 they, with Henderson and Haggard,[9] advanced the mucus plug theory to explain not only massive collapse of the lung, but also pneumonia. They regarded massive lobar collapse as the condition from which post-operative pneumonia developed, and advocated the treatment of pneumonia and, of course, of massive collapse by the inhalation of an oxygen mixture containing 5% carbon dioxide. They reported that in several hundreds of cases this treatment had given excellent results.

Occasional cases have been recorded in which acute massive collapse of the lung has followed upon injection of the bronchial tree with lipiodol. A point of considerable interest noted in several of these cases has been the length of time between the injection and the collapse. In cases reported by Jacobaeus[10] ten to fifteen minutes elapsed between the injection and the recording of the skiagram. In a case reported by Scott Pinchin and Morlock[11] in 1931 the collapse occurred within three minutes. It will be remembered that when Coryllos and Birnbaum produced massive pulmonary collapse in animals by plugging a bronchus with a balloon, the collapse was not complete till six hours later. In the cases of massive collapse following so quickly after the injection of lipiodol Jacobaeus put forward the view that reflex spasm, involving the walls of the bronchioles, was the only way in which the lung could be so rapidly emptied of its air.

In 1938 Jones and Burford,[12] of New York, reported four cases in which death followed quickly after the administration of cyclopropane anæsthesia. Autopsy revealed massive atelectasis of one or both lungs. Their paper should be of great interest to anæsthetists. As in the cases reported by Bergamini and Shephard, the fact is here again emphasized that acute massive collapse of the lung may be a cause of sudden death on the operating table. Further interest is attached to the fact that in the subjects examined *post mortem* by Jones and Burford no evidence was found of gross bronchial obstruction. They suggest that the mechanism of the collapse in these cases is related to the presence of rapidly absorbable oxygen and cyclopropane in certain areas of the lung, where, as a result of long-continued shallow respirations, little ventilation is occurring; so that patches of atelectasis may develop without an obstruction in any part of the bronchial tree.

Atelectasis or Massive Collapse Associated with other Pulmonary or Intrathoracic Lesions.

While most interest in massive collapse of the lung has been associated with the post-operative condition, massive atelectasis is of considerable interest to the physician for other reasons. With the occlusion of a bronchus by a growth atelectasis develops, as shown by the limitation of movement on the affected side and by the displacement of the heart. The slow development of atelectasis may be an important sign of bronchial carcinoma. A large shadow or opacity may be seen in the skiagram of the lung field, when later a *post mortem* examination may reveal a very small bronchial carcinoma

completely occluding a bronchus and causing massive collapse of portion of a lung.

The literature contains many references to the frequency of occurrence of massive atelectasis in cases of pulmonary tuberculosis. Sanes and Smith,[13] writing in 1937 on the subject of massive pulmonary atelectasis following bronchial obstruction in tuberculosis, stated that a review of the literature indicated that the incidence of massive atelectasis in pulmonary tuberculosis was more frequent than its diagnosis. They suggested that in cases associated with hæmorrhage its presence was sometimes overlooked because of the physician's pardonable reluctance to submit such patients to the ordeal of a complete physical and radiographic examination. In most reported cases bronchial obstruction was the usual cause, and specific obstructing causes have been variously noted, such as enlarged lymph glands and peribronchial fibrosis, coagulated blood, fibrinous bronchial casts and caseous material. These authors also support a view put forward by Hennell,[14] in 1931, that certain cases in which a diagnosis of unilateral fibroid tuberculosis was made were cases in which an atelectatic lung had failed to reaerate itself and had become fibrotic.

These views upon the frequent incidence of atelectasis as a complication of pulmonary tuberculosis have surprised me considerably, and I must confess that I have not seen this condition in the tuberculosis department of the Royal Prince Alfred Hospital. Even admitting that I have not looked closely for it, it would seem reasonable to expect that it would be discovered on radiographic examination.

From this cursory review of the very extensive literature on this subject it has been shown that the condition of massive collapse of the lung occurs in many varied disorders; and it would seem that there is not always some common ætiological factor and that probably the mechanism whereby it is brought about is not always the same.

Symptoms and Physical Signs of Post-Operative Massive Collapse of the Lung.

Post-operative massive collapse of the lung may present itself clinically in different ways, which are probably determined by the suddenness with which bronchial plugging occurs, by the size of the affected bronchus, by the speed and degree in which mediastinal shift occurs, and, lastly, by the subsequent infection which may supervene in the collapsed area of lung.

Symptoms.

In the cases hitherto reported in which death has occurred during or very soon after operation, it has been noted on several occasions that although the operation was of a simple character and the anæsthesia not complicated, respiratory failure has developed abruptly, followed later by failure of the circulation, resulting in death within short periods, varying from about three to twenty minutes. These sudden deaths, however, appear to be rare, and the condition usually manifests itself from a few hours to as long as seven days after some abdominal operation.

Pasteur originally described two types of this condition: the acute type and the latent type.

Acute Post-Operative Massive Collapse.—Acute post-operative massive collapse may again present itself in two forms: (i) The patient may present symptoms which suggest a catastrophe, such as pulmonary or coronary embolism or pneumothorax. The onset is characterized by initial dyspnœa of great intensity and suddenness, which is often accompanied by pronounced cyanosis and collapse. (ii) In the commoner type the symptoms resemble those of acute pneumonia. The pulse and the respiratory rates are raised. The temperature may rise to 39·5° C. (103° F.). Pain in the chest on the affected side is frequent at the onset, and usually there is a cough accompanied by expectoration of viscid sputum which later becomes mucopurulent but does not contain blood. The presence of blood, altered or unaltered, demands a different diagnosis.

Latent Post-Operative Massive Collapse.—In latent post-operative massive collapse there may be a complete absence of symptoms, although the physical signs are usually well developed. Such cases may easily escape detection if the patient's back is not examined.

Physical Signs.

In massive collapse of the lung movement on the affected side of the chest is greatly diminished, while movement on the unaffected side is exaggerated. Dulness to percussion is noticed over the affected side, usually the base of the right lung; the dulness corresponds with the area of collapsed lung and may extend up to the clavicle. In one type of case the breath sounds are diminished or absent; in another type loud tubular breathing, usually without râles, may be heard, and bronchophony and pectoriloquy may be pronounced. The difference in physical signs is probably dependent upon the degree of patency of the bronchi. It is readily seen that the physical signs in the lungs are those commonly attributed to pneumonic consolidation.

By far the most important physical sign of massive collapse is the displacement of the heart towards the affected lung. This is the cardinal physical sign of the condition, and it cannot be diagnosed with any certainty in the absence of this sign. Displacement of the heart, however, will not occur in cases of bilateral collapse or if the mediastinum is fixed. Moreover, it is possible that with only small areas of partial collapse in a lung no great displacement of the heart will occur.

When recovery occurs and the collapsed area of lung begins to reexpand, the heart will gradually move back to its normal position. As the air enters the reexpanding lung, râles may be heard over the affected area.

The pulmonary signs may vary from time to time also, because if the intrabronchial secretion is not too viscid it may spill from its original site when the patient's posture is changed, and air may enter the previously involved areas.

Radiographic Appearances.—X ray examination affords a valuable addition to physical examination in confirmation of the diagnosis. The site and extent of the collapse are indicated by a dense homogeneous shadow. The density of this shadow is very much more pronounced than that of the shadow given by a partially collapsed lung seen in a case of pneumothorax. The skiagram will also confirm the displacement of the mediastinum and the heart shadow towards the affected side. The trachea may be displaced if an upper lobe alone is involved. The diaphragm is elevated on the affected side. There is compensatory emphysema of the unaffected lung.

Differential Diagnosis.

The diagnosis of the condition is more likely to be made by the medical man who is looking for it. If the possibility of its occurrence is always suspected after an abdominal operation, the characteristic physical signs, especially the displacement of the heart, will enable the diagnosis to be made. X ray examination, whenever possible, will confirm the diagnosis.

Where the onset is sudden and severe, attended by gross dyspnœa, cyanosis and collapse, pulmonary embolism will be closely simulated. In acute massive collapse the heart will be displaced and blood will be absent from the sputum.

Pneumothorax is a common mistaken diagnosis. This diagnosis may be suggested in right-sided collapse by the hyper-resonance over the healthy lung associated with the disappearance of the dulness of the heart. Here the true nature of the lesion will be revealed on auscultation by the presence of exaggerated breathing.

Probably the greatest difficulty arises in distinguishing acute massive collapse from pneumonia. There can be no doubt that some of these cases at least have frequently been regarded as instances of post-operative lobar pneumonia on account of the pronounced physical signs of consolidation. The only real method of distinction between these conditions is determination of the position of the apex beat. If this cannot be made clinically X ray investigation should be carried out whenever possible. The subsequent course of the illness may give helpful information, as the general symptoms of massive collapse are invariably less severe than those of pneumonia. In a doubtful case the course of the temperature may give useful information. In a case of collapse the temperature rises steeply at first to 38·9° or 39·5° C. (102° or 103° F.) and after eight to twelve hours gradually falls to normal. The reappearance of fever after a short fall in temperature usually indicates the onset of inflammatory changes in the lung.

The displacement of the heart may lead to an erroneous diagnosis of dextrocardia or of dilatation of the heart as an explanation of the misplaced cardiac area.

Finally, the pain in the chest and the physical signs may lead to a diagnosis of pleurisy with or without effusion.

In some very acute cases the diagnosis will be strongly suggested by the fact that spontaneous recovery may occur in a spectacular fashion after a severe fit of coughing, which may partially, if not completely, remove the obstructing material.

Treatment.

Prophylactic Treatment.

Under the heading of prophylactic treatment may be mentioned the simple precautions that are usually observed, such as the recognition of respiratory infections and their elimination before operation, or the postponement of the operation in the presence of a respiratory infection whenever it is possible.

The question of premedication before the anæsthetic will be adequately dealt with by my colleague; but Grey Turner[15] may be quoted in this regard: he "did not think it was a good thing for a patient to arrive in the anæsthetic room in a sort of comatose condition, and it was certainly very serious for such a patient to lie in the same state for hours after returning to the ward".

Given a quick recovery from an anæsthetic, the posture of the patient should be changed frequently, and fixed or unnatural postures should be avoided whenever possible. The patient may be lowered from a sitting to a lying position or rolled from one side to the other. Coughing may be encouraged sometimes by the administration of a little morphine to relieve the pain associated with it. Tight bandaging of the upper part of the abdomen or of the thorax has been condemned by many, although the bandage can hardly be dispensed with when a patient is coughing or vomiting. The patient may be encouraged to take several deep breaths to expand the lung bases several times daily.

Treatment of the Developing Condition.

When the signs indicate that collapse is already occurring, more vigorous methods are necessary in order to cause the lung to reexpand. In the early stages of collapse the inhalation of oxygen containing 5% carbon dioxide is of great value. The opinion has been expressed that the use of carbon dioxide as a hyperventilating agent at the end of the operation, in order to wash out the anæsthetic, is undoubtedly of value; but the patient should be allowed to fill his lungs with air rather than with oxygen. In the later stages of collapse the hyperventilation produced by carbon dioxide may produce the reverse of the desired effect, in driving the bronchial plug further on.

Bronchoscopic aspiration cannot be advocated lightly as a general rule. In many cases simple methods of treatment, such as change of posture,

are sufficient, or spontaneous recovery may occur after a fit of coughing. There will usually also be a natural reluctance to submit a very sick patient to this procedure unless adequate facilities and the necessarily skilled bronchoscopist are available. On the other hand, there seems ample evidence in the literature to suggest that bronchoscopic aspiration, well carried out, may entirely relieve massive collapse of the lung, and do so rapidly, thus possibly avoiding the risks of subsequent infection in the collapsed lobe.

When acute collapse is associated with pronounced shock and collapse, stimulants of various types may be used; another suggestion which has been made is the introduction by means of a pneumothorax apparatus of 100 to 200 cubic centimetres of air into the pleural cavity on the affected side, in order to raise the intrapleural pressure.

Conclusion.

My own experiences have not qualified me to make any original contribution in regard to massive collapse of the lung. With interest, but partly in despair, I have asked some of my senior colleagues of their experiences. A customary reply has been that one, or perhaps two, of these cases have been seen by an individual observer. Usually the diagnosis has been suggested when some obvious post-operative pulmonary complication has spontaneously cleared up after a spectacular bout of coughing, or when the presence of a large area of apparent consolidation has been found soon after operation without the usual toxæmia associated with pneumonia.

In most of the cases which have thus been described to me, X ray examination had not been carried out, either because facilities were not available or because the patient was too ill.

The fact that X ray facilities have not been used to any extent in the diagnosis of post-operative pulmonary complications immediately becomes apparent when an attempt is made to obtain even one X ray film typical of this condition of massive collapse of the lung.

Reference to the record departments of our metropolitan hospitals usually reveals that there is the necessary pigeon-hole for case records of atelectasis; usually one, sometimes several such histories, may be found. In one hospital the one case of atelectasis recorded occurred in a stillborn fœtus.

Further inquiry also usually reveals an absence of any very complete or satisfactory method of recording post-operative pulmonary complications of any kind. There would appear to be a great necessity for some special interest and study in this direction, especially when premedication is becoming a routine procedure and many new types of anæsthetics are being used.

If a death should occur from massive collapse of the lung during an operation it is probable that the condition will be overlooked, because such a case is usually reported to the coroner and no *post mortem* examination may be made.

So far also as I have been able to discover, the indication has very rarely arisen for a bronchoscopic examination to be made for this condition in any of the metropolitan hospitals.

It would seem to me, on considering the evidence, that one of two conclusions is open to us. Firstly, we can say that massive collapse of the lung is very rare and of little practical importance. We may even think that for some obscure reason it does not occur in Sydney. Or, alternatively, we may decide that we shall look for atelectasis or massive collapse of the lung, each of us—surgeon, physician, anæsthetist and bronchoscopist. We shall suspect every pulmonary complication after an abdominal operation. We shall have X ray photographs taken of the lungs in doubtful cases of post-operative pneumonia. And when we have done all this and recorded our findings over several years, somebody else will probably then be able to read a more instructive paper on this subject.

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THE TREATMENT OF SOME COMMON DISORDERS ENCOUNTERED IN INDUSTRIAL PRACTICE.

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THE following conditions have been chosen as the subject of this paper because they are frequently encountered by the industrial surgeon and the

general practitioner, and because they do not respond to treatment as readily as they should. In this latter respect I am afraid I cannot offer some miraculous method of curing any or all of them overnight, but I shall describe the methods that have yielded the best results in the greatest number of cases, and the basis of them all is just common sense.

Traumatic Arthritis of the Fingers.

Traumatic arthritis of the fingers is nearly always the result of a direct blow to the affected joint, be it metacarpo-phalangeal or interphalangeal. The joint very soon becomes swollen and it is painful from the beginning; it is hot to the touch, tender and limited in all its movements. The swelling is mainly periarticular, but there is always a certain amount of synovial effusion. This latter point is somewhat difficult to demonstrate in these small joints. The skiagram invariably reveals no abnormality, but in some instances small chips from the articular surfaces can be seen.

Treatment.

It is well known that fingers do not bear immobilization well, nor do they respond to passive movements or forceful manipulations. Why this should be, nobody has properly explained. As the treatment of traumatic arthritis in other joints is rest and, in some neglected cases, manipulation, some other method must be employed in similar conditions of the fingers. There is one exception to this—the child. If anyone under the age of twelve years (this is an arbitrary figure) should sustain a traumatic arthritis of a finger, it is quite safe to apply a splint, but only for two weeks at most.

In the adult there is every possibility that a permanently stiff finger will be produced by the use of immobilization. The safest and surest way of treating this condition is by self-massage with the aid of hot soapy water.

The method is so simple that it will not appeal to the imagination of many patients, not to mention practitioners. The patient is instructed to fill a basin with water as warm as he can bear it. He then makes it soapy by the use of sufficient soap, so that he can massage the affected part without producing any appreciable friction of the skin; during the process it is essential to exert a moderate amount of pressure on the finger. This massage is continued for twenty minutes or more, and during the whole period efforts are made to move the affected joint. The treatment is carried out two or three times daily, and as the tenderness abates the period can be prolonged. For the first few days the procedure is best carried out under supervision, so that it is certain that the patient understands what is necessary. The restoration of movement in stiff fingers depends entirely on the patient; the most that can be done, short of surgical intervention (which will not be dealt with here), is the placing of the fingers into the optimum position of rest under an anæsthetic, followed by encouragement of the patient to work from that position. This last manœuvre refers rather to a generalized stiffness of the fingers, such as that following a septic infection of the hand, which has been immobilized for several weeks. In these cases the use of self-massage with hot soapy water frequently yields good results.

Another method that may be employed is the use of moulding clay that has been warmed; the patient is given a ball of clay the size of a tennis ball, and he is instructed to squeeze it into different shapes. Warmth is essential in these procedures, for it brings about a hyperæmia with a consequent ability of the tissues to yield and stretch.

If a chip fracture is present it may be ignored and the treatment continued as if it were not present. Some surgeons prefer to remove all fragments; but I think the larger ones only should be removed, for the presence of the operation wound delays the institution of treatment of the arthritis.

With the method of treatment I have outlined you will still find a number of patients suffering from these injuries taking a considerable time to recover; however, once all tenderness has disappeared it is quite safe for the patient to return to work, as use will increase the range of movement.

There is an exception to every rule, and with these adult patients it is this: occasionally arthritis resulting from injury will be too acute for self-massage to be begun immediately. In these cases a splint or some similar device will be needed; but it should not be retained for more than a week at most.

The advantages of this method of treatment are: firstly, its superiority over other methods in common use for traumatic arthritis of the fingers; secondly, its simplicity; and thirdly, its inexpensiveness.

Teno-Synovitis of the Wrist.

Teno-synovitis is a misnomer for the condition which it denotes, a condition which is so common in the lower part of the forearm; it is in reality peritendonitis or, if it is situated higher in the forearm, perimyositis. If the patient who complains of this condition can be persuaded to allow the surgeon to cut down on the swollen, tender and painful part, the pathological changes will be found to be confined to the areolar tissues surrounding the tendons and muscle bellies. These tissues, the perimysium and the peritenon, are œdematous and jelly-like, and contain fibrin deposits; the synovial tendon sheaths are not altered and there is no evidence of *synovitis sicca*. The primary changes are in the muscle tissue; there is a depletion of glycogen followed by acute degenerative muscle changes, and the interstitial deposits of fibrin in the perimysium and peritenon are the cause of the diagnostic crepitation on movement of the affected muscles. Furthermore, careful examination will disclose the fact that the swelling, pain, tenderness and crepitation extend higher up the forearm than any tendon sheaths known to anatomists.

The most likely causes of glycogen depletion of muscles are: (i) unaccustomed and prolonged use of certain muscles, (ii) ordinary use following trauma. The muscle groups most frequently affected in the region of the wrist are the radial extensors, the extensors and long abductor of the thumb, and the flexors of the fingers.

Treatment.

As there is always a local increase in the pH to a high acid level, and as there is nearly always an associated oxaluria, it is a good plan to administer large doses of potassium citrate. I have seen several patients with this condition respond rapidly to this treatment when other methods have failed. It is therefore a wise plan to test the urine in every case.

The main object in treatment is complete immobilization of the affected muscles. This can be achieved by any method the surgeon favours. There is no sense in immobilizing the fingers if the wrist extensors and the thumb muscles only are involved; but if the finger muscles are affected, they should be immobilized on a long cock-up splint which is appropriately carved to maintain the fingers in the position of rest. In no circumstances should the fingers be immobilized in the straight position, even for the ten days or so that are necessary to bring about a resolution in the muscles and surrounding tissues. If plaster is used, the same principle holds good; no joint should be immobilized in a position of strain. It is as well to point out that failure to bring about a quick response to treatment is due to the fact that the thumb is not immobilized when the muscles on the lower part of the dorsum of the forearm are involved. Ten days' immobilization will be sufficient in the great majority of cases. After the removal of the splints the only treatment necessary is restoration of movements by active use before the patient returns to work. The employment of massage or various forms of heat or motion does not bring relief, but aggravates the condition; and this is only to be expected when the pathology of the condition is considered. The application of adhesive strapping will be effective in only the mildest cases.

Olecranon Bursitis.

Simple Bursitis.

Simple bursitis may be due to constant irritation from pressure and friction, or it may follow a single injury in the nature of a blow. In either case the bursa is filled with a straw-coloured fluid; and in the former type, which may be termed the chronic type, there may be no symptoms beyond some discomfort when full flexion of the elbow joint is attempted. In the other type, which can be termed the acute type, the symptoms are generally more pronounced.

Treatment is the same in both types; aspiration under rigid aseptic precautions, with the subsequent application of a pad under firm pressure, will prevent the recurrence of the fluid in most cases. If this method fails there should be no hesitation in dissecting the bursa out of the surrounding tissues. This form of treatment will be necessary in all chronic cases in which the bursal wall is unduly thickened.

Hæmorrhagic Bursitis.

Hæmorrhagic bursitis always follows a direct blow, and at times it is very painful. If the injured limb is seen early and if the bursa should contain blood, the contents will need aspiration, so that the formation of fibrous nodules on the walls of the sac will be prevented. After this the application of firm pressure over a pad will prevent the further escape of blood and serum. On the other hand, if the condition is seen only after the formation of fibrous nodules, the only form of treatment that will give permanent relief is excision of the bursa along with the fibrous thickenings. This latter state of affairs is easily diagnosed by palpation of the irregular and tender thickenings in the region of the bursa.

Suppurative Bursitis.

As in the case of a septic joint, the route of infection in suppurative bursitis can be from the surface by direct implantation, via the blood stream, via the lymphatics or by direct spread from a neighbouring focus. The only sure way of dealing with a bursa that has become infected is to open it widely by means of a generous incision as soon as the presence of pus has been diagnosed. The small incisions generally made in this condition are very inadequate, and a three-inch incision in the long axis of the arm should be the minimum. The next desideratum is the use of infrequent dressings; once adequate drainage has been instituted, Nature will do the rest if she is left alone.

If some residual stiffness of the elbow joint should follow a suppurative olecranon bursitis, do not allow any massage to the joint or anything in the shape of passive movements; if the movements are going to return, active use is the only therapy of any value, while the other methods frequently cause traumatic arthritis. It is well known by those who employ manipulative surgery that the elbow joint, along with the joints of the fingers, responds very badly to manipulation in any shape or form.

Paronychia and Eponychia.

Paronychia and eponychia can prove real stumbling blocks to treatment, and the difficulty of obtaining resolution of the infective process in most instances is due to overlooking of the presence of a subungual abscess. In its simplest form infection in the area round the base of the nail shows itself as a "pus blister"; this readily clears up after removal of the roof of the blister. On the other hand, if a subungual abscess is present the treatment will be very prolonged unless the

lesion is dealt with appropriately. Its presence may be difficult to recognize at times; but if there is a change in colour of the nail, especially of the lunula, to a pale yellow or even to a bluish-red, the diagnosis is reasonably certain. The treatment of the condition, once an abscess has formed under the nail, is removal of sufficient of the base of the nail to produce proper drainage, and of all of the nail substance that is acting as a foreign body. Very often not enough nail is removed, and that which is left still acts as an irritant. If this adequate removal of the nail does not bring about a reasonably rapid resolution, the surgeon should consider some other cause than simple infection—syphilis, melanoma, epithelioma *et cetera*.

At times, when one of these infections has been neglected, an incision may be needed to prevent spread of the infection up the finger. These incisions should be made in such a situation that they penetrate the reflected layer of the epithelium and not the nail bed itself; in other words, they should extend proximally from the lateral edge of the nail bed and should never be made over the nail bed itself. If the latter mistake be made it is very likely to be followed by an unsightly deformity of the nail or its base. Some surgeons recommend an incision on each side of the nail bed; but the indications for such a procedure are very few. If more attention were paid to adequate nail removal the need for any of these incisions would be practically non-existent.

Reports of Cases.

A FATAL CASE OF INFECTIVE ENDOCARDITIS DUE TO NEISSERIA FLAVA.

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THE following case history and bacteriological notes record an example of fatal infective endocarditis, due to an inhabitant of the naso-pharynx normally regarded as non-pathogenic. *Neisseria flava* was isolated from the blood stream and from the heart valves at *post mortem* examination.

Clinical Record.

Miss E.H., aged twenty years, was admitted to hospital on August 19, 1938. Her past history included a first attack of acute rheumatic fever at the age of six years, with recurrent attacks of rheumatism, the last at the age of fourteen years. In January, 1938, at the age of nineteen years, she first complained of feeling languid and easily exhausted. On July 1, 1938, she had eleven teeth extracted under nitrous oxide and oxygen anæsthesia. Forty-eight hours later she complained of anorexia, nausea and attacks of pain in the back and wrists, accompanied by a temperature varying between 37·7° and 38·4° C. (99·8° and 101° F.).

Examination on her admission to hospital revealed a slightly enlarged heart; a presystolic thrill and presystolic and systolic murmurs were noted at the mitral area. Radiological examination of the heart revealed enlargement of the left auricle. The systolic blood pressure was 160 and the diastolic pressure 116 millimetres of mercury. Examination of the lungs revealed no abnormality, and no enlargement of the liver or spleen was detected. The fingers were not clubbed; although no petechial hæmorrhages were observed in the skin, these were later found in the conjunctiva. Examination of the blood revealed that the erythrocytes numbered 2,850,000 per cubic millimetre and the leucocytes 10,800 per cubic millimetre, 75% being polymorphonuclear neutrophile cells; the hæmoglobin value was 50%. The urine contained numerous red blood cells. An attempt at culture from the blood, made on August 20, yielded no growth.

The temperature persistently varied between 37·3° and 38·9° C. (99° and 102° F.). Some nausea and vomiting were present, and on September 7, three weeks after her admission to hospital, the patient had a rigor. From a blood culture made that day a Gram-negative coccus was grown. Further rigors followed, and on September 17 a blood culture again yielded a Gram-negative coccus. Death occurred on September 20.

Post Mortem Examination.

A *post mortem* examination was made eight hours after death. The heart was slightly enlarged and mitral and aortic valvulitis was present. The mitral valve orifice was stenosed, and, superimposed on the distorted cusps, were firm, adherent, brownish-yellow vegetations (Figure I). No vegetations were present on the auricular wall. The spleen was moderately enlarged and contained many infarcts; some infarcts were also found in the kidneys. Cultures made at the *post mortem* examination from the vegetations on the heart valves yielded a pure growth of the same Gram-negative coccus.

Photograph of heart, showing vegetations on the mitral valve.

Bacteriological Findings.

It will be noted that the first attempt at blood culture yielded no growth; but the difficulty of obtaining cultures from the blood in cases of subacute bacterial endocarditis is well known. Two subsequent successful attempts at blood culture and the isolation of the organism *post mortem* established the nature of the infecting organism.

The organism isolated had the following characteristics. Morphologically the organisms were spherical or oval cocci arranged singly, in pairs or in clumps, and varying somewhat in size. They were Gram-negative and non-motile. Incubation on ascitic agar or agar for twenty-four hours at 37° C. resulted in a growth of round, convex, discrete, greenish-yellow translucent colonies, 0·5 to 2·0 millimetres in diameter, with smooth glistening surface and entire edge, of butyrous consistency and fairly easily emulsifiable. Incubation on agar at 22° C. resulted in no growth.

Broth cultures, incubated at 37° C. for twenty-four hours, showed a granular deposit and slight turbidity. The organisms gave the following biochemical reactions: acid but no gas was produced in glucose and maltose; there was no change in mannite, lactose or sucrose.

The greenish-yellow colour of the colonies and the growth on ordinary agar place this organism in the group known as *Neisseria flava*. As it is recognized that there are considerable variations in the cultural characteristics of the organisms of this group and that no satisfactory classification into types is as yet possible (Topley and Wilson, 1936), we have refrained from assigning this organism to any of the types that have been described.

Discussion.

Two points of interest arise in this case. The first is the fact that *Neisseria flava* has been shown to be the sole infecting organism in a fatal case of subacute bacterial endocarditis. Goldstein (1934) described a case due to an organism of the *pharyngis* group, and Libman (1920) stated that occasionally subacute bacterial endocarditis was caused by *Neisseria flava*, but gave no details of any cases. No other references to *Neisseria flava* in this connexion could be found in the available literature.

The second point is the question of the route by which this organism, a normal inhabitant of the naso-pharynx, gained access to the blood stream. The observations of Okell and Elliott (1935) that following on tooth extraction a transitory bacteriæmia may occur in patients with or without oral sepsis are of interest in this connexion. It is almost certain that in this case extraction of a large number of teeth allowed access to the blood stream of organisms present in the mouth and naso-pharynx, notably of *Neisseria flava*, which organism became established in the heart valve already damaged by rheumatic disease.

Summary.

A fatal case is recorded of subacute bacterial endocarditis, due to *Neisseria flava*, an inhabitant of the naso-pharynx in normal human beings.

Acknowledgement.

The authors wish to express their appreciation of Dr. Eric Cooper's interest and advice in the preparation of this note.

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Reviews.

DIETETICS FOR THE CLINICIAN.

"WHAT shall we eat?" and "When?" are questions likely to interest most of us. For sick people the questions sometimes call for a good deal of thought, and a newly published little book, "Dietetics in General Practice", by Dr. Leslie Cole, will be found a great help to the general practitioner when he has the responsibility of caring for patients whose dietary programme is a matter of some importance.[1]

In these recent years there has been a spate of talk about the dependence of national prosperity on proper feeding of the people. Numerous special inquiries have been made by experts into nutrition problems, and such slogans as "Drink More Milk" and "Eat More Fruit" vie with others, such as "Beer is Best", to gain the public favour.

It is an old story that wrong feeding can do a lot of harm, and now that their causes are recognized, such maladies as rickets and scurvy are rare.

The gross defects arising from long-continued malnutrition are fortunately less common in these days than formerly, yet even in our sunny Australia subclinical rickets is sometimes seen. In modern times science has been so helpful, and Nature, aided by science, so lavish that none in the world should go hungry. There is enough food for all.

Some may complain that the modern interest in food has been carried too far and that the setting up of a score or more of pseudo-scientific "systems" of dieting has been the result. This may be so, yet, if food fads are so common, the trouble can be corrected only in one way, and that is by making generally available to the people reliable information on adequate and well-balanced diets. Here is the field for the general practitioner. Coming into contact with people in their homes, he is able to advise on the everyday problems of planning diets for sick people, diets for pregnant and nursing women, and diets for children.

This is one of the family doctor's responsibilities, and Dr. Cole's book will be found a valuable help in enabling the doctor to fulfil the task. Wrong feeding may cause disease, while the correct food is often the best medicine in the prevention and treatment of illnesses.

Dr. Cole deals first with the underlying physiological basis of suitable diet for the healthy, and discusses some of the common dietetic faults. He gives suitable emphasis to the importance of sound dental hygiene and the effect of diet on this problem. He sets out in a clear way the types of diet suitable in various gastro-intestinal disorders and also the modifications in diet necessary in a wide range of health disorders.

It is a small book, but it is packed with sound advice, embodying the result of the extensive researches of recent times. For the research worker on nutrition problems the book may have little use; for the general practitioner it is well worth the money asked for it.

VITAMINS AND VITAMIN DEFICIENCIES.

THE first of a projected seven volumes on vitamins and allied factors, intended by the author to provide a "comprehensive synopsis" of contemporary knowledge in that field, has been published.[1] With an already voluminous and overwhelming literature on the subject, which grows in complexity almost daily, Dr. Harris's work is very opportune, and satisfies a deep need among students and other workers who in their search for information cannot hope to probe the enormous mass of original literature.

With his own ample experience of personal research on vitamins, Dr. Harris is well fitted to appraise the work of others, and in the first of these volumes he has undoubtedly succeeded in his object to present a concise yet sufficiently detailed picture of the results of modern research complete with bibliography.

The historical introduction is a very fair account of pioneer work on vitamins, and vitamin B_1 is considered in all its aspects—clinical, chemical, biochemical and physiological.

Features of the book worthy of particular mention are the inclusion of statistics as to the incidence of avita-

[1] "Pocket Monographs on Practical Medicine: Dietetics in General Practice", by L. Cole, M.A., M.D., F.R.C.P.; 1938. London: John Bale, Sons and Curnow Limited. Foolscap 8vo, pp. 162. Price: 6s. net.

[1] "Vitamins and Vitamin Deficiencies", by L. J. Harris, Ph.D., Sc.D., D.Sc., F.I.C.; Volume I: Introduction and Historical, Vitamin B_1 and Beri-Beri, with a foreword by F. G. Hopkins, O.M., F.R.C.P., F.R.S.; 1938. London: J. and A. Churchill Limited. Large crown 8vo, pp. 218, with 50 illustrations. Price: 8s. 6d. net.

minoses in various regions, a consideration of "conditioned deficiencies", hypovitaminosis and its detection, alternative methods for the estimation of the vitamins and their relative merits, the mode of action of the vitamin, and an enumeration of the more newly recognized vitamin factors.

Dr. Harris also stresses the point that a wide intellectual gap separates the mere arbitrary use of anti-scurvy, anti-beri-beri or anti-rhachitic food adjuncts administered empirically, and the full recognition that these diseases are primarily and directly caused by simple deficiencies in the diet.

The book is recommended to all students and even the specialist as an admirable summary of the facts available today, with a bibliography fully up to date.

THE STORY OF VITAMINS.

FROM time immemorial diagram has been the means *par excellence* for the communication of an idea. The eye is a big help in the comprehension of either fact or principle, and modern educationists are insisting more and more upon the value of visual education as a method of instruction. Miss Gregory, in her book "A B C of the Vitamins",[1] has enlisted the visual instruction method to present the story of the vitamins. Her book is a comprehensive survey in charts of our present knowledge of the vitamin substances. In it she has concentrated scientifically accurate data into a form in which the facts can be grasped at a glance. Every aspect of the subject—history, deficiency diseases, geographical distribution of deficiency diseases, the chemistry of the vitamins and their function in the body—is graphically and concisely presented, with an excellent bibliography for those who wish to pursue the subject further.

This book does not purport to be original in the sense of reporting results of research. Its value lies in the new method of scientific writing. As the author states in her preface, this method has been used in the past to present small groups of physiological facts to illustrate books and articles. Such illustrations have been repeatedly published to an extent that points the need for this particular type of presentation. Some of the charts are very simple and easily read; others require careful study if they are to be completely understood.

Because it so successfully combines clarity, brevity and scientific accuracy, this book is recommended either to the layman or to the scientific student who wishes to grasp quickly the essentials of the story. It should prove particularly useful as teaching material to the physiological chemist, the nutrition worker, the practising physician and the medical student.

NUTRITION AND DIET.

IT is a significant and hopeful sign that so much thought is now being given to constructive medicine, and particularly to the part that nutrition must play in its extension. As evidence of this growing interest in nutrition, the seventh edition has now appeared of "Nutrition and Diet Therapy", a text-book of dietetics, by Fairfax T. Proudfit.[2] In this new edition the material has been completely revised and recent advances in nutritional science have been incorporated.

Miss Proudfit has considered in detail, first, normal nutrition and its practical application, and, secondly, the construction and preparation of therapeutic diets. In the dietetic section there has been added an entirely new series of laboratory lessons designed to coordinate the different chapters of lecture material. In the diet therapy section the laboratory procedures emphasize the modifications of the normal diet necessary to meet special pathological conditions. A useful appendix is attached of food values and equivalents.

Primarily designed to fit into the accepted scheme of nursing education in the United States of America, this volume provides useful teaching material in both the principles and the practice of nutritional science.

[1] "A B C of the Vitamins: A Survey in Charts", by J. Gregory, M.S., with a foreword by W. H. Eddy; 1938. London: Baillière, Tindall and Cox. Royal 4to, pp. 106, with 56 charts. Price: 12s. 6d. net.

[2] "Nutrition and Diet Therapy: A Textbook of Dietetics", by F. T. Proudfit; Seventh Edition, completely rewritten and reset; 1938. New York: The Macmillan Company; Australia: Angus and Robertson Limited. Demy 8vo, pp. 931, with illustrations. Price: 17s. 6d. net.

Notes on Books, Current Journals and New Appliances.

ANÆSTHESIA AND SURGERY IN GENERAL PRACTICE.

FROM *The British Medical Journal* comes the third volume of articles on treatment in general practice, reprinted from the issues of that journal between July 17, 1937, and June 25, 1938.[1] This volume deals with anæsthesia and surgery. For those who do not remember the articles as they appeared, we should point out that in the anæsthesia section the choice of anæsthetic in general practice is discussed and there are articles on recent advances in anæsthesia, the physiology and pharmacology of anæsthesia, the stages and signs of general anæsthesia, the depth of anæsthesia for various operations, basal anæsthesia, local anæsthesia, analgesia in midwifery, general anæsthesia in dentistry, endotracheal anæsthesia, spinal anæsthesia, preoperative treatment and medication, post-operative treatment, risks of anæsthesia, apparatus and equipment, anæsthesia and the law.

The section on surgery has, as the editor of *The British Medical Journal* points out in the preface, been designed for the country doctor who is thrown largely on his own resources—and there are many of these in Australia. There is no need to give a list of all the chapters in this section, but we welcome contributions on the use and abuse of antiseptics, on the treatment of wounds and on such apparently simple subjects as burns and scalds, blood transfusions, saline infusions, abrasions and contusions, and boils and carbuncles. Even those who bind their copies of *The British Medical Journal* may be glad to have these articles in one compact volume, which is of convenient size and well produced. We hope that this volume will meet with the same success as its predecessors have enjoyed.

A DICTIONARY OF BIOLOGY.

THE third edition of Henderson's "Dictionary of Scientific Terms" has been published.[2] This book deals with pronunciation, derivation and definition of terms in botany, zoology, anatomy, cytology, embryology and physiology. It will be useful to the student of medicine in his preclinical years and to those who are interested in biological science. It is not intended to replace a medical dictionary, but may be used with advantage in conjunction with one. The type face has been well chosen and the printing is excellent.

[1] "Treatment in General Practice. Articles Republished from The British Medical Journal. Volume III: Anæsthesia: Surgery"; 1939. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 113, with 60 illustrations. Price: 10s. 6d. net.

[2] "A Dictionary of Scientific Terms. Pronunciation, Derivation, and Definition of Terms in Biology, Botany, Zoology, Anatomy, Cytology, Embryology, Physiology", by I. F. Henderson, M.A., and W. D. Henderson, M.A., B.Sc., Ph.D., F.R.S.E.; Third Edition, revised by J. H. Kenneth, M.A., Ph.D., F.R.S.E.; 1939. London and Edinburgh: Oliver and Boyd. Demy 8vo, pp. 395. Price: 16s. net.

The Medical Journal of Australia

SATURDAY, JULY 22, 1939.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

MEDICAL CERTIFICATION.

THE work of a medical practitioner does not end when he has examined a patient, made a diagnosis and prescribed certain forms of treatment. Even when the sick man has recovered the practitioner cannot always regard the affair as complete. The patient may be employed by a business firm or association of persons, he may be a member of a friendly society lodge or some other body which arranges for contract medical practice, he may be insured under the provisions of the *Workers' Compensation Act* or by a policy effected directly with an insurance company. In these circumstances the medical practitioner will almost certainly be called upon to give to the patient a certificate or certificates setting out the nature of his illness or injury and the length of time for which he is likely to be disabled; he will also probably be asked at a later date to state that the patient has sufficiently recovered to be able to resume his usual occupation. In more than one of the Australian Branches of the British Medical Association the question of certification has recently been raised both in regard to certificates issued about the illness or injury of persons receiving payments during their incapacity from a government department, and in regard to friendly society lodge certificates.

Medical certification has been discussed in these pages on previous occasions, and from recent events it would appear that medical practitioners need to be reminded from time to time of their duties and responsibilities in this matter. Sometimes government departments make requests of medical practitioners for details of patients' histories which they should not be asked to supply. On the other hand, friendly societies and other bodies associated with contract medical practice often require, with complete justification, details which the medical practitioner, in writing his patient's certificate, does not give. Medical certificates may be certificates of fact or they may be an expression of opinion. In other words, the medical attendant is often asked to state for how long, in his opinion, an employee will be unable to follow his usual occupation. The difference between facts and opinions must be constantly kept in mind. That a patient has broken his leg is a fact; it is also a fact that he is suffering from a febrile disturbance, commonly known as "influenza". Whether the broken leg was occasioned by the patient's work may not be determinable by the medical attendant. Should he find the patient at the foot of a ladder in his workshop, he may justifiably conclude that the injury was inflicted in the course of work. A statement as to how long the unfortunate patient will be disabled from a fractured leg or from "influenza" is a matter of opinion. This is self-evident and should not need emphasis, yet the need for emphasis is repeatedly being made obvious. In this regard mention must be made of the not uncommon practice of the post-dating of certificates; such a course of action may be adopted by a medical attendant out of sheer good nature to save the patient an extra journey to the surgery; or the attendant may do it to save himself trouble on a future occasion. If it should happen that the patient is unable to visit the practitioner at a later date the certificate should bear the date of the day on which it is issued and should be worded something as follows: "In my opinion Mr. X will be able to resume duty on such-and-such a date." It is unfortunately not unknown for medical practitioners to give a lodge certificate to a lodge secretary, whom he regards as a reliable

and good fellow, on behalf of a lodge member whom he has not examined in connexion with the disability about which he is writing a certificate. This is a most reprehensible practice. Again, a lodge patient, or any other patient, may present himself at the surgery with the statement that he has been in bed for three days with "influenza" and ask for a certificate. The proper course for the medical practitioner to adopt is for him to write: "Mr. X informs me that he has been in bed for three days with 'influenza'; he is now able to resume his work." If he thinks that the patient's general appearance is in keeping with his statement, the medical attendant may add this to the certificate. But he must in no circumstances give a certificate as to fact unless he has by his own observation made certain that the patient's statements are true. When a medical practitioner engaged in contract practice writes a certificate for a patient on his "list" he should be just as careful and take just as much trouble as he would in the case of a private patient. Sufficient detail should be given to allow the officials of the contracting organization to realize the gravity of the injury or illness. To state that a patient is suffering from "injury to the hand" is useless. The extent of the injury and its nature—contusion, laceration, fracture and so on—should be stated in detail. By the omission of these details medical practitioners lay themselves open to accusations of carelessness; and he who is careless in small will be careless in greater matters.

As far as medical certificates to government departments are concerned, circumstances have recently arisen in which a government department has asked medical practitioners to give directly to the department certificates setting out in full the nature of a patient's illness and other details connected with the patient's health, and this without the consent of the patient having been obtained. Requests of this kind should be resisted by the members of the medical profession. If the patient's consent is obtained there can be no objection; neither can there be any objection if the certificate is given to the patient for transmission to the department. If the patient comes to the practitioner from the department which is seeking a report, the practitioner should make it clear to the patient that the report is to be sent to the department. Even collaboration of the medical attendant with a departmental medical officer without the knowledge and consent of the patient is wrong, and should not be considered. There should be no misunderstanding in this matter; the medical attendant is the repository of the patient's secrets, whether the secret has been revealed by the patient or merely observed by the medical attendant. Nothing must be countenanced which will in any way undermine the confidence of the patient in his medical attendant.

A medical practitioner may be known by the type of certificate which he issues. Government departments, insurance companies, friendly societies and employers in any community, particularly in a small community, soon learn the identity of medical practitioners on whom they can rely. And patients are not slow to realize that a medical practitioner is determined to be fair and just in his certification of their illnesses and accidents. One who is meticulous and accurate in such a matter as certification will, they argue, be just and careful in affairs which possibly concern them more deeply. Truly "by their certificates shall ye know them".

There is another point which should be emphasized. The General Medical Council of Great Britain has issued to medical practitioners a salutary warning which might, and probably would be, adopted by medical boards in Australia, guided as they are in so many other matters by the "G.M.C." The warning runs as follows:

> Any registered medical practitioner who shall be shown to have signed or given under his name and authority any such certificate, notification, report or document of a kindred character which is untrue, misleading or improper, whether relating to the several matters above specified or otherwise, is liable to have his name erased from the medical register.

We do not believe that Australian medical practitioners would wilfully issue false certificates, but it is obvious that care must be taken in the giving of certificates relating to alleged facts. A careless practitioner might be called upon to defend himself on the grounds of an information laid against him because of an incorrect or misleading certificate.

Current Comment.

A VIRUS PNEUMONIA IN INFANTS.

THE list of virus disorders is steadily enlarging, and now a virus pneumonia secondary to epidemic infections in infants has been described by E. W. Goodpasture, S. H. Auerbach, H. S. Swanson and E. F. Cotter.[1] The authors state that they have for some years been seeking evidence of virus activity in routine autopsies, and particularly in examinations of the respiratory tract. It is an established fact that the respiratory system is affected by several virus diseases in the human subject. However, it was only comparatively recently that the authors met with evidence that could be reasonably interpreted as indicating activity of a virus in the respiratory system, except in certain instances in the lungs of children. In these cases "protozoa-like cells" were observed, identical with those sometimes found in the parotid glands of infants and of some lower animals. In the case of guinea-pigs and mice these have been shown to have been caused by viruses. Goodpasture and his associates now record five instances of a specific type of respiratory infection in infants, of whom the oldest were two and a half years of age. The lesions of the respiratory tract indicated that, apart from associated bacterial infection, a specific virus determined the phenomena observed. Characteristic intranuclear inclusions present almost exclusively in epithelial cells of the trachea and bronchi, of their mucous glands and of the alveoli, were essential details. Epithelial necrosis and ulceration of the tracheal and bronchial mucosa followed, giving a curious appearance to the lesions. The first example was noticed in 1931, after measles. The next one (in 1936) was that of an infant two weeks old, who had had no previous infection. The three other cases occurred in the spring of 1938; two followed measles and the remaining one after pertussis. Thus in four of the cases the infection followed measles or pertussis.

The infection is severe and may terminate fatally, either primarily or from bacterial invasion. The five cases are all reported in detail, and Goodpasture and his associates consider that the respiratory tract in all presented a fairly uniform picture, but that no other organ or system, in any of the cases, exhibited lesions of importance. In one instance examination of the parotid gland did not reveal protozoa-like cells. In the case which followed pertussis there was evidence of encephalitis, but no virus inclusions were noted in any cells of the nervous system. In all the cases the trachea and bronchi showed hyperæmia, with a suggestion of ulceration in places. The lungs varied as to evidences of consolidation superficially, but in each case sections disclosed scattered areas of consolidation. A characteristic feature was the presence of hæmorrhage in the lung, which was either isolated or situated about areas of inflammatory consolidation. In the centre of each of the consolidated areas was a softened opaque spot or line which consisted of pus surrounded by a thin zone of pneumonic consolidation, which was often red itself or was surrounded by hæmorrhage. The central areas grossly resembled small bronchiectatic abscesses or bronchi and bronchioles filled with exudate. In all cases the trachea and larger bronchi exhibited similar microscopic changes. The surface was entirely denuded of ciliated epithelium and was in part ulcerated. The rest was covered by a thin epithelial surface, often only one cell in thickness. The ulcerated areas sometimes were covered by fibrin. A characteristic feature consisted of scattered areas of necrosis in the mucous glands, involving one or more lobules. Just below the basement membrane of the mucosa there was an exudate of polymorphonuclear leucocytes, most abundant at areas of ulceration. Elsewhere it was mixed with mononuclear cells. Polymorphonuclear leucocytes were present in and on the epithelial layer. In the smaller bronchi and bronchioles there was often total destruction of epithelium. The lumina were filled with cellular exudate and necrotic débris. Bacteria were often demonstrable in them. Extending beyond and about the bronchioles, there were patches of pneumonitis involving alveoli. The exudate sometimes was predominantly hæmorrhagic. In places it was fibrinous and cellular. At times there was an aspect suggesting the early lesions of tularæmia. Alveolar walls were implicated in the necrotizing process, which extended from the bronchioles. Bacteria were demonstrable in the bronchiolar lumina and in the areas of necrosis.

The specific feature, according to Goodpasture and his colleagues, was the presence of intranuclear inclusions, which were almost entirely restricted to epithelial cells. In one instance a few fibroblasts surrounding an ulcerated bronchus contained similar but smaller bodies within their nuclei. The specific nuclear inclusions were slightly acidophilic structures, separated from the nuclear membrane by a clear zone. Sometimes the inclusion filled the nucleus and then, as in *herpes simplex*, it seemed to be slightly basophilic. The affected nuclei and sometimes the whole cell were slightly enlarged, but not to so great an extent as were the cells affected by the parotid virus. The affected cells rapidly underwent necrosis, which was the cause of the extensive ulceration. Experiments undertaken by Goodpasture and his colleagues eliminated, in their opinion, *herpes simplex* virus as the cause of the manifestations observed. They further considered that the affected epithelial cells of the respiratory tract did not resemble those of "inclusion disease". They state that if the virus of these cases is related to the latter it must be an unusually virulent strain that has become altered so as to induce a different kind of lesion. Experimental inoculation of infected lung into rabbits, mice, opossums, chicken embryos and a *Macacus rhesus* monkey failed to establish the infection.

[1] *The American Journal of Diseases of Children*, May, 1939.

Goodpasture and his associates consider that there are indications that this virus infection is becoming increasingly important as a secondary invader of the respiratory tract in Tennessee following acute epidemic diseases and tending to initiate fatal bacterial invasion of the lungs. Accordingly they stress the importance of a constant watch for its occurrence.

THE USE OF PREPARATIONS OF CREOSOTE IN PULMONARY TUBERCULOSIS.

CONAN DOYLE, in one of those early romances in which he so successfully anticipated the modern literary addiction to detection of crime, gave the famous Sherlock Holmes a good start in the race to justice by making one of the criminals step in a pool of spilt creosote. The pungency of odour which led the detective's canine lieutenant across London on that occasion has made an even greater impression on the sensitive nostrils and palate of many a patient. Creosote is not very often prescribed today, but a generation ago it was given freely to the majority of sufferers from "weak chests", whether this euphemism denoted pulmonary tuberculosis or a chronic respiratory catarrh due to an unrecognized sinusitis. Today creosote is still widely used, but like many stars of yesterday it is now assigned a minor role, and is merely regarded as an adjuvant or is used in the form of an ingredient in certain proprietary preparations whose basis is cod liver oil. Those who have had a wide experience with this drug will recall how small doses are usually tolerated well, but increasing or prolonged dosage may cause gastric irritation. Many of us must have wondered if creosote had genuine virtues, whether it contained some specific which discouraged the tubercle bacillus, and whether it was directly excreted by the epithelium lining the respiratory tract. The most recent work on this subject is by Edwin J. Fellows, who has been studying calcium creosotate.[1]

Previous pharmacological work has shown that this drug is chemically only slightly different from the creosote from which it is prepared, and it has been employed instead of the original creosote in the treatment of pulmonary tuberculosis, bronchiectasis and suppurative pneumonitis. Fellows draws attention to the fact that the Council on Pharmacy and Chemistry of the American Medical Association has omitted all creosote preparations from the list of "New and Non-Official Remedies" because it was believed that the only basis for their use was purely empirical. He therefore undertook an investigation to determine whether any modification of sputum could be observed in patients taking calcium creosotate. The patients chosen for observation were suffering from pulmonary tuberculosis, as these were found to produce more constant quantities of sputum than those suffering from other cavitating diseases of the lungs. The quantity and type of sputum were noted, and in addition an estimation was made of the volatile phenol content of the sputum before and during periods of administration of the drug. In this way it could be ascertained whether the creosote was excreted into the air passages, while evidence of its absorption was gained by estimation of the volatile phenol content of the urine. Some attempts were also made to detect the presence of phenolic substances in the air expired by laboratory animals to which calcium creosotate had been administered. The observations on patients were continued over three months and adequate controls were employed. The amount of calcium creosotate given was one tablet of 0·26 gramme three times a day, increased gradually so as not to cause nausea. It was not found possible to exceed a dose of two tablets three times a day. It is interesting to read that although some patients tolerated the drug well, after one month of administration many patients became nauseated, so that it became necessary to suspend the drug. This recalls the clinical experiences with creosote referred to above.

The volume of sputum from the patients being treated and from those used as controls was found to be remarkably constant and no evidence of any variation in volume or character of sputum could be found following the administration of the drug. No evidence could be obtained of excretion of creosote in the sputum, nor could any phenolic substances be traced in the expired air of laboratory animals receiving calcium creosotate. There seems no doubt that the drug was absorbed, because the urine of the patients and animals under review contained large amounts of volatile phenols. An attempt was made to find out whether the patients experienced relief from cough, or more effortless expectoration, or if their appetites improved, but no evidence of any such change was obtained. It would seem, therefore, that if there is any virtue in creosote, it has evaded scientific proof up to the present, and it is perhaps unlikely that the waning reputation of the drug will be revived.

[1] *The American Journal of the Medical Sciences*, May, 1939.

THE VITAMIN CONTENT OF MUTTON BIRD OIL.

DURING the last few years mutton bird oil (from the local petrel *Puffinus tenuirostris*) has been sold in Victoria for the same purpose as cod liver oil. The oil is obtained from the stomach of the young bird at the end of the nesting season, and it has been regarded as a reliable source of vitamin *A*, following the work of Malcolm (1926), who found that "mutton bird oil is one of the richest known natural sources of vitamin *A*", and of Carter and Malcolm (1927), who found, however, that the vitamin *A* content of the oil was variable. These biological experiments were carried out in the very early days of vitamin *A* estimations, before the modern physical and chemical methods were perfected, and consequently these early results cannot be expected to be accurate.

In view of the importance attached to mutton bird oil, therefore, the vitamin *A* content of various specimens has recently been estimated by William Davies[1] in Melbourne by non-biological methods, namely, the Carr-Price process. Davies used the Lovibond tintometer to estimate the intensity of the blue colour in the non-saponifiable fraction, and the measurement of the intensity of absorption at 328mμ of a solution in cyclohexane (Davies and Field, 1937).

Estimations have been made with both commercial and fresh non-commercial samples, and it appears that the vitamin *A* content of the oil is of the very low order of 0·005% (four blue units), and therefore virtually useless as a medicinal source of vitamin *A*, even before it has been subjected to the commercial refining processes which destroy the vitamin. Jowett and Davies (1938) have shown that the liver oils of common local fish, such as the snapper, shark and barracouta, usually contain between 0·5% and 4% of vitamin *A*, that is 100 to 800 times the concentration in mutton bird oil; while Hines and Callaghan (1937) in Queensland have shown that ordinary mullet liver oil is of considerable value in this respect. It would therefore be much better to exploit these oils as a vehicle for vitamin *A* than to continue the use of mutton bird oil. One difficulty presents itself, however, in this regard, at least in the commercial exploitation of mullet liver oil in Queensland, that there is already a keen demand for mullet gut in Brisbane, and mullet livers could be obtained only at a price too high to justify the commercial preparation of the oil. The difficulty of separating the small liver from the gut is also a commercial problem.

Mutton bird oil was assayed also for vitamin *D* by the prophylactic biological method of Hume, Pickersgill and Gaffikin (1932) by the use of albino rats. It is reported that the oil contains "the very low value of five international units per gramme" and cannot be compared with cod or halibut oils as a vehicle for vitamin *D*.

Finally, Davies has pointed out that the oil is of little use as a food, as it is mainly a liquid wax rather than a true fat.

INFANT MORTALITY IN INDIA.

THE problems of infant mortality are much the same the world over. Differences are mainly in the degree of difficulty that the problems present. Everywhere the greatest obstacle is ignorance. This can be countered to some extent by legislation. But in some countries, such as India, no suitable legislation exists. Also many other measures must be undertaken if infant mortality is to be greatly reduced. B. Muktha Bai has made a study of the causes of infantile deaths in India. His paper on the subject is worthy of notice.[2] He first points out that, as India has a very high birth rate, the high infant mortality rate is apt to pass unnoticed. He stresses the necessity for the enforcement of registration of births and deaths to enable proper estimates to be formed. It has been suggested that a high rate of infant mortality is one of Nature's methods of checking over-population. But, as Muktha Bai points out, it is an uncivilized and unscientific measure. Further, it must be remembered that the causes that operate to kill some will maim or debilitate many others. "The problem of infant mortality can never be dissociated from the problem of infant morbidity." It has also been suggested that reduction of the infant death rate would merely result in the survival of weaklings. But this has been proved incorrect by Ballard's investigations in England. Ballard noted that weakly children were actually less susceptible to summer diarrhœa, for example. Muktha Bai divides the first twelve months of life into neonatal (the first month) and post-natal (the remaining eleven months) periods. The most important cause (direct or indirect) of death in the neonatal period is prematurity. In many cases no cause other than prematurity is reported. The next most important cause is infection. In India there is greater risk of infection than in many other places, because of the dirty habits of the *dai* (native midwife), who will cut the cord with any instrument that is sharp enough. "The social custom of considering the mother and infant as something untouchable has to be blamed here along with the public authorities who do not take action against the *dai*." Muktha Bai places birth injury third on the list of causes of neonatal death. He regards overcrowding as a grave source of disease, hence mortality, during the post-natal period. Of 216 infants who had died, 201 had lived in one-room tenements occupied on an average by 4·8 persons per room. Pneumonia was the most important single cause of death in this period. He stresses the importance of natural feeding in the prevention of diarrhœa. Indian mothers used to think that suckling was a sacred duty. Modern ideas tend to dispel this belief, and women are more inclined to feed their infants artificially. As this tendency becomes more pronounced, so will diarrhœa increase. Artificial feeding also sometimes leads to starvation, which comes fourth in the list of causes of deaths; for "ignorance, poverty and poor health prevent the mother from giving enough food and care to her children". Muktha Bai stresses the danger of many social customs and superstitions.

Superstitions are many. Many diseases are thought to be due to some evil spirit and no attempt is made to cure it in any scientific way. Many refuse to get their infants vaccinated in spite of knowing the benefits.

Medical practitioners interested especially in infant welfare would be repaid for the time spent on the perusal of Muktha Bai's paper. Perhaps the most important lesson it teaches is that there can be no excuse for the continuance of an evil. An evil thing should be eliminated if for no other reason than that it is evil.

[1] *The Australian Journal of Experimental Biology and Medical Science*, March, 1939.
[2] *The Indian Medical Gazette*, June, 1939.

Abstracts from Current Medical Literature.

DERMATOLOGY.

The Value of Patch Tests.

JOHN G. DOWNING (*The New England Journal of Medicine*, November 3, 1938) discusses the value of the use of patch tests in dermatology. The early part of the paper is concerned with a description of the history and methods of application of patch tests, and the grades of reaction are set out according to the classification of Bloch. But the most detailed portion of the paper deals with results and interpretations. He quotes a paper by Wise and himself as stating that the value of the tests is limited to *dermatitis venenata* and drug eruptions of an eczematous type, such as those due to mercury or arsphenamine. A warning is also given against the use of primary skin irritants in the carrying out of the tests. Elimination of the offending substance is stated to effect a cure; but attempts at desensitization are not recommended. Next, numerous fallacies in interpretation of the results, "false positive" and "false negative" results, are reviewed. Among these is mentioned the liability of certain test substances, such as oil, tar, trichophytin *et cetera*, to produce acneiform lesions. Very delayed responses, as described by Ingram, are also mentioned. These are considered to be new sensitizations. "False positive" results may be due to substances that injure normal skins. Here the use of a control is stressed. A person may respond to some substance with which he suspects contact but which he actually does not handle in the circumstances in which the test is made, though previous contact may have occurred. Tests made too near the affected area or in areas where complete recovery has not occurred may give rise to "false positive" reactions. "False negative" reactions may occur through too great a dilution of test chemical, through selection of an unsuitable area, or through a state of hyposensitivity of the patient at the time of testing. Also it is generally impossible for physical agents, trauma or vital activity to be reproduced. The author next discusses the value of some experimental work which has been performed. This includes comparisons between the reactivity of infants and of adults, variations in dilution, differences in time and the use of different areas, comparisons between normal and atopic persons, and, of particular value from a practical viewpoint, the use of patch tests to estimate the possibility of forecasting the likely onset of arsenical dermatitis in patients requiring therapy with the arsenobenzol derivatives. The latter investigations have shown that patch tests are valueless in this respect. A final detailed survey of treatment and an evaluation of patch testing from a more general viewpoint, especially that of industry, are made. The author points out the impossibility of knowing the composition of the test substances in all cases, the importance of the tests being carried out by the physician, and the necessity for the actual conditions of contact to be reproduced as far as possible. The danger of the production of new sensitivities by over-testing is stressed; and in this respect it is of great value to supervise industry and avoid the hazard rather than to perform patch tests upon numerous workers and remove them from their work. "False positive" reactions may cause unnecessary expenditure for the elimination of suspected hazards, while the real hazard remains. "Pseudo-negative" reactions may give a false sense of security. Many substances may create a definite industrial hazard without being at all suitable for patch-testing. Patch tests may also prove a misleading factor to the lay mind in medico-legal cases. The author concludes by stating that patch tests in themselves have no diagnostic value, but that in conjunction with a carefully taken history showing definite exposure to the test material, and with a characteristic test eruption, the prevention of further exposures should result in a cure. He considers that, properly employed, the patch test is superior to scratch and intracutaneous tests.

Toxic Dermatoses following Sulphanilamide Therapy.

JAMES WILLIAM TEDDER (*Archives of Dermatology and Syphilology*, February, 1939) surveys the literature and states his own views on the subject of toxic skin manifestations following the use of the sulphanilamide group of drugs. There would appear to be little unanimity concerning the distribution or most common type of eruption in the experience of most workers, except that many of the rashes encountered appear to be precipitated or aggravated by sunlight. In the author's opinion the individual eruptions were of three general clinical varieties: (i) the most frequent type, maculo-papular or morbilliform rash, of rather limited distribution, precipitated or aggravated by sunlight; (ii) widely distributed rashes, ranging from macules and papules to œdema and urticarial wheals with occasional coalescence—probably the most irritating type; (iii) purpuric rashes—the most rare. The author discusses several cases of the sunlight-induced variety. The rashes soon subsided when the drug was withheld. In two cases the patient withstood ultra-violet irradiation without recurrence; but in another case a general eruption was precipitated by this means. Patch tests and passive transfer tests were, however, without result. The conclusions reached by the author are as follows. The first and most common eruption is precipitated by sunlight. It is not due to high blood sulphanilamide content, nor to sensitivity to the drug ordinarily, but to a combination of internal factors, such as possible abnormal porphyrin metabolism, lowered resistance due to the disease and irritation due to the sun. The result is a localized dermatitis which may be complicated by eruptions at distant points. The second type of eruption is that in which true sensitivity occurs—a sensitization dermatitis. The third type is due to poor toleration of or saturation with sulphanilamide—a true toxic dermatosis, after which the patient will tolerate further sulphanilamide only when excretory functions become normal once more or when the dosage is properly adjusted.

The Reaction of the Stools in Skin Disease.

R. AITKEN (*The British Journal of Dermatology and Syphilis*, January, 1939) discusses briefly, with some short case records, the relationships between certain skin diseases and the reaction of the fæces. He commences by reiterating the well-known connexion between certain dermatoses and toxic absorption from the bowel. He points out that not infrequently colonic lavage helps to relieve the condition, but that such relief is temporary and that relapse is common after cessation of the lavage. The author, working on the assumption that the normal human stool should be faintly but definitely acid, has investigated the reaction of the stools in a number of cases of different types of skin eruption of a chronic nature. Types of disease quoted by the author include *pruritus ani*, chronic urticaria, light dermatitis, lichenification and neurodermatitis. In all the cases described the stools, tested by litmus, were found to have an alkaline reaction. If the reaction was rendered acid the clinical condition of the patient was greatly improved or cure was effected. The regime is a simple one. The patient is advised to drink one pint of buttermilk daily and to take a dessertspoonful or a tablespoonful of lactic oats. In the author's experience it may take two to three months for the stool reaction to change. When the patient is in hospital colonic lavage at the beginning of treatment is recommended.

Sex Hormones in Acne Vulgaris.

IAN D. RILEY (*The British Journal of Dermatology and Syphilis*, March, 1939) discusses the value of testosterone propionate in the treatment of *acne vulgaris*. He discusses first the relationship of the disease to the development of sexual activity and to the female menstrual cycle, about which relationship opinions differ. Several workers were unable to obtain satisfactory results by using anterior pituitary hormone or œstrin. The author next describes briefly the isolation and biological action of

testosterone and testosterone propionate. The main subject matter of the paper, however, deals with a report of twenty patients with acne treated with the hormone and ten other patients used as controls. The latter were given 0·5 cubic centimetre of distilled water monthly by subcutaneous injection. The hormone was exhibited by injection over a period of three months in gradually increasing weekly doses up to 50 milligrammes per week. Total dosage averaged just over 300 milligrammes. The conclusion reached was that the treated patients showed slight but definite improvement as compared with the ten controls, but that the results were not greatly superior to simpler routine measures in the small number of patients treated.

UROLOGY.

Incontinence of Urine in Women.

C. GOMEZ (*Journal d'Urologie*, October, 1938) describes a number of successful results with the reparative operation of Marion for stretched or torn vesical sphincter resulting from injury during labour. A transverse incision is made through the mucosa of the anterior vaginal wall just behind the external urethral meatus. The mucosa is dissected up and the posterior edge retracted well so as to expose the tissues beneath. A de Pezzer catheter has previously been placed in position; its mushroom head enables the surgeon to define the exact position of the internal meatus. The most lateral part of the exposed fibromuscular tissue on each side of the urethral meatus are brought together under the urethra by means of transverse mattress sutures of silk or thread. Reinforcement over this layer is effected by apposition of the anterior edges of the *levatores ani* (ischio-clitoridal) muscles. The de Pezzer drain is left in position for about twelve days. Should the patient experience difficulty in natural micturition on its removal, intermittent catheterization is performed for a day or two.

Double Ureter and Bifid Ureter.

C. COLOSIMO (*Urologia*, October, 1938) says that the terms "double ureter" and "bifid ureter" may be used as corresponding respectively to complete and incomplete reduplication of the ureter. Such bifidity and doubling combined reached a percentage of 3·33 in 1,500 skiagraphic examinations of the urinary tract by excretion urography. The anomaly was found to be more frequent in the female sex, the proportion being 68%. The reason for this preponderance has not been determined. The most frequent types were unilateral doubling and unilateral bifidity. In double ureters the two canals cross, the exception to this rule being very rare. The vesical openings of the double ureters constantly followed the law of Weigert, which states that the ureter draining the upper (supernumerary) portion of the kidneys opens into the bladder in an inferior and medial relation to the other orifice. As a rule inspection of a double kidney reveals no sign of demarcation between the two portions. In a few cases, however, a more or less definite sulcus is present. In one case the author observed that the division was so complete that there appeared to be two kidneys on the same side; moreover, the vascular supply was distinct. The anomaly was almost always accompanied by some urinary tract disturbance, due to the anomalous vascularization, the mechanico-dynamic disturbance and the generic predisposition of anomalous organs to morbidity. Pain is a symptom of greatest importance. In about one-half of the cases the lesions were of such a grade as to be clearly demonstrable skiagraphically. Symptoms most commonly arose when the patients were between the ages of twenty and thirty years. Although it has been stated above that there is a tendency to morbidity in the supernumerary or anomalous portion, it was observed in three cases that the disease process established itself in normal territory, leaving the anomalous portions free. In all, the author found that morbid processes were about equally distributed between the upper and lower pelves. Some authors, however, have found that dilatation and other lesions are most frequently confined to the upper pelvis.

Ascending Ureteric Tuberculosis.

AUGUSTO BARELLA (*Urologia*, October, 1938) discusses, with reference to clinical notes and autopsy specimens, a very interesting and important type of renal tuberculosis, in which advanced renal tuberculosis is present on one side and the lower portion only of the opposite ureter becomes infected from below. The ureteric papilla becomes tuberculous by direct spread of the secondary tuberculosis of the bladder; the uretero-vesical junction becomes incompetent and allows reflux of urine containing tubercle bacilli. A certain number of inches of the lower end of the ureter thus become tuberculous while the rest of the same ureter, together with the kidney above it, remains healthy. The appearance in the excretion urograms is characteristic and is familiar to all urologists; on one side is a functionless or very diseased kidney with rigid ureter. The bladder is of the usual small spherical type, of permanent contraction, while the opposite kidney and upper part of the opposite ureter appear as normal shadows, the lower portion of the opposite ureter being, however, rigid and slightly dilated. In the particular case under consideration death occurred from advanced pulmonary tuberculosis before the diseased kidney could be removed. In an autopsy specimen comprising the bladder with both ureters and one kidney, a tuberculous pyonephrosis is seen on the right side and advanced tuberculosis of the right ureter and the bladder is present. The tuberculous process has spread up the lower half of the left ureter; but the upper half of the right ureter, as well as the left kidney, is quite normal to the naked eye. Complete freedom of the latter portions from tuberculosis was proved microscopically. In the matter of diagnosis in such cases a warning is issued against an immediate conclusion that bilateral renal tuberculosis is present, for the patient is deprived of the possibility of cure by nephrectomy.

Expectation of Life after Ureteral Transplantation.

H. WADE (*Edinburgh Medical Journal*, February, 1939) has performed transplantation of the ureter on sixty patients, and in addition has traced a number of patients operated upon by his senior colleagues. In one such case the operation took place over thirty years ago and the patient remains without any clinical or urographic evidence of renal damage. He concludes that when the operation is performed as a last resort the immediate mortality is in the region of 50% and the expectation of life is very short, owing to the extension of the original disease. Nevertheless, in the successful cases the intolerable pain and frequency of micturition are relieved. On the other hand, when the operation is undertaken for congenital defects, injuries *et cetera* in otherwise healthy patients, the immediate mortality is low (about 5%), and those who recover may anticipate a long and useful life, unimpaired by physiological or social inconvenience.

Decompression in Chronic Retention of Urine.

COMPLETE and immediate evacuation of the chronically distended bladder, as recently advocated by Creevy, is strongly deprecated by J. R. Dillon (*The Journal of Urology*, February, 1939). He points out that, although the theory that sudden relief of intravesical pressure is the cause of renal œdema and hæmorrhage has not been proved, neither has it been disproved. It is well known that 90% of patients with chronic urinary retention will not be endangered by sudden emptying of the bladder; but this does not warrant the risking of the lives of the remaining 10%. In addition to the risks of renal failure and hæmorrhage, Dillon considers that infection more easily invades a kidney which is the site of gross congestion. This is illustrated in the cases cited, and it is emphasized that, although the kidney may be able to resume its function, the general condition of such patients is so poor that they are unable to resist a virulent infection.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on June 29, 1939, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. G. BARRON, the President, in the chair.

Massive Collapse of the Lung.

DR. S. V. MARSHALL read a paper entitled "Anæsthesia and Pulmonary Atelectasis" (see page 124).

DR. W. A. BYE read a paper entitled "Massive Collapse of the Lung" (see page 129).

DR. M. P. SUSMAN said that he hastened to agree with Dr. Marshall that the anæsthetic was only one factor in the production of post-operative atelectasis. He was in agreement with Dr. Bye concerning the relative rarity in Australia of massive collapse of the lung; he had been looking very hard for the condition during the previous four or five years. He had encountered two cases only during 1939; the patients were a man suffering from a perforated duodenal ulcer and a youth with acute appendicitis. Both recovered. Dr. Susman had been struck with the lack of toxæmia and the disparity between the radiological and clinical signs on the one hand and symptoms on the other. In a series of thoracic operations for empyema, drainage of lung abscess, lobectomy for bronchiectasis, and thoracoplasty for tuberculosis, Dr. Susman had not encountered this complication; further, it had not occurred in a series of 76 operations for phrenic nerve paralysis for pulmonary tuberculosis. When much purulent sputum was present, as in bronchiectasis or lung abscess, Dr. Susman used bronchoscopic drainage immediately before the operation; he believed that this measure reduced the tendency to post-operative atelectasis. Although massive collapse of the lung was generally considered a post-operative complication, Dr. Susman pointed out that it could occur before operation; in those circumstances it was an indication of the patient's poor condition. Dr. Susman quoted the case of a man, aged thirty-six years, who had a perforated duodenal ulcer. When Dr. Susman saw him his pulse rate was 136 per minute, he was sweating and cyanosed, dulness to percussion at both bases was present with bronchial breathing, and he had peritonitis. He died three days after operation. At the *post mortem* examination the lower lobes of both lungs were found to be airless; no pneumonia was present. That condition had been present before the patient was subjected to operation. Dr. Susman said that it was doubtful whether obstruction was the main cause of post-operative massive collapse of the lung; but it was well known that obstruction could cause atelectasis apart from operation. Dr. Susman then showed slides illustrating collapse of an area of the lung following obstruction of a bronchus in two cases. In one of these the condition was proved by biopsy to be a carcinoma of a bronchus. The skiagram taken after instillation of lipiodol showed the obstruction very clearly. In this case Dr. Susman had inserted a radon container through which the patient was able to breathe, and in a few days the lung had reexpanded.

DR. A. S. WALKER said that he thought that the papers and discussion had proved the wisdom of the choice of the subject. No doubt a considerable number of those present felt a little uneasy about the condition under discussion. Dr. Walker said that about ten years earlier he had encountered a very pronounced case of massive collapse of the lung; he had then studied the subject and had been on the look-out for examples of the condition, and yet when Dr. Bye had asked him about it, Dr. Walker's experience was extremely limited. Dr. Walker thought that the disorder was uncommon in Australia, but that lesser degrees of collapse of the lung were not uncommon. It might be also that practitioners had not the facilities that they should have for examining patients in whom massive collapse of the lung might occur. It might be better if patients who had complications after operation were subjected to more careful scrutiny. Dr. Walker agreed with Dr. Bye that it was impossible to guess the position of the heart with much accuracy. Dr. Walker had recently seen another case of massive collapse; he thought that he had recognized it perhaps because Dr. Bye had spoken to him about the subject. The lesion had not been very pronounced, but it had one extraordinary feature: the collapse had occurred a week after operation. Referring to the ætiology, Dr. Walker said that it was understandable that a bronchial obstruction could cause pulmonary collapse; it was possible also that trauma might cause collapse, bringing in some of the ætiological factors that Dr. Bye had suggested; but it was hard to understand why a patient who was doing extremely well after an abdominal operation should have a pulmonary complication a week later. Whenever Dr. Walker had been asked to see patients who had suffered pulmonary complications after operation he had tried to bear in mind the possibility of pulmonary collapse and pulmonary embolism as well as that of bronchopneumonia, a diagnosis which was sometimes loosely made. He considered that pulmonary embolism was more common than pulmonary collapse. Dr. Walker said that he wished to draw attention to the fact that not only the signs but also the symptoms of massive collapse of the lung simulated bronchopneumonia. The one sure sign was displacement of the heart. It was possible for the medical attendant to be put off because the patient might produce a little blood or blood clot in the sputum.

Dr. Walker said that he was not competent to criticize Dr. Marshall's paper; however, in the days when he used to give insufflational anæsthetics he had felt a little uneasy when after an operation under "intratracheal" ether the patient was "over blown" and breathed very little. Dr. Walker thought that this uneasiness had probably been well founded. Modern methods of anæsthesia, although having great advantages, needed to be very carefully handled. Dr. Walker said again that he considered both papers presented a very good review of the literature on the subject. On reading the available literature some years ago he had noted that it was concerned largely with experimental work. He had perhaps looked on this with a certain amount of suspicion, because conditions that could be produced in laboratory animals did not always entirely resemble those occurring in human beings. However, more work should be done. Dr. Walker thought it important to find out how a slight injury, particularly to the thoracic wall, could cause atelectasis, and why a patient doing well after an abdominal operation should suffer massive collapse of the lung a week later.

PROFESSOR C. G. LAMBIE said that he had found the papers of great interest. He had been impressed by the excellent historical review of the subject given by Dr. Bye. Dr. Bye had put the whole matter in a nutshell. The two things really known about the disorder under discussion were that it occurred under conditions of immobility of the chest and of obstruction. However, that raised the question of the cause of the obstruction. Out of the numerous causes one or two that figured in the literature had not been mentioned. Papers had been published in which evidence had been adduced to show that the incidence was greatest among patients supposed to be allergic. It would be interesting to know whether the leucocytosis which was not infrequently present was associated with a relative increase in the number of eosinophile cells. Professor Lambie went on to refer to the infective hypothesis. He pointed out that plugs causing obstruction in bronchi had been removed by aspiration, and when examined had been found to contain not only mucus but pneumococci and fibrin, and the fibrin was present in proportion to the pneumococci. In this connexion it was interesting that sometimes a rise in temperature occurred to 103° or 104° F. Some authors held that lobar pneumonia arose in a similar way from infection through the walls of the bronchi. Pneumonia could be produced experimentally by instillation of pneumococci into the bronchi, not by their injection into the blood

stream. Professor Lambie then commented on the physical signs of massive collapse of the lung. He said that in pneumonia the breath sounds sometimes disappeared or were very weak; this was believed to be due to plugging of the bronchi with mucus. Collapse of the lung was a condition supposed to be due to blockage of the bronchi, and yet in its presence bronchial breathing was heard. Professor Lambie wondered what was the explanation of the apparent paradox. Referring to the part played by anæsthetics in the occurrence of massive collapse of the lung, Professor Lambie said that he felt incompetent to discuss them. He took it that the danger associated with the administration of readily absorbable volatile anæsthetics was related only to the occurrence of blockage or immediately after operation. It could not apply to collapse occurring eighteen or thirty-six hours afterwards.

DR. H. C. R. DARLING said that the subject was one in which he had been particularly interested for a number of years, and he thanked the speakers for their excellent summaries. In his opinion collapse of the lung occurred in two forms: an endemic form, "that was always with us", and an epidemic form, that manifested itself when medical attendants forgot their physiology and pharmacology. Dr. Darling said that he restricted his remarks to pulmonary collapse occurring subsequent to ether anæsthesia. The induction and maintenance of anæsthesia depended upon the absorption into the blood stream of a given amount of ether. Factors influencing absorption were concentration of the vapour and pulmonary ventilation. If the respiratory exchange was diminished, the concentration of vapour necessary to obtain surgical anæsthesia must be increased. After abdominal operations the respiratory exchange was greatly diminished, by as much as 50% or 60%, though it was held by some that 80% was the figure. With ordinary ether anæsthesia the endemic type of pulmonary collapse might occur and the patient did not experience much discomfort. Referring to the epidemic type of massive collapse, Dr. Darling said he had witnessed two epidemics. The first occurred about 1908 to 1912. Dr. Darling was at that time working at University College Hospital, near Middlesex Hospital, and the subject of massive collapse was on everyone's tongue. It had taken him twenty-five years to discover the cause of this epidemic. Heroin (dimorphine hydrochloride), in doses of from one-twelfth to one-eighth of a grain, had temporarily replaced morphine as the routine post-operative medicament. It was not realized that this drug had almost a specific action as regards the abolition of the cough reflex. Dr. Darling went on to say that the next epidemic of massive collapse of the lung that he encountered occurred when he was unwise enough to try preliminary narcotization with "Avertin" prior to the administration of ether; the result was five cases of apneumatosis in one week. Dr. Darling then set about discovering the reason why this should have been. The reason was bound up in the question of respiratory exchange. He said that very little note was taken of the amplitude of respiration, which was the most important factor in respiratory exchange. The patients on whom "Avertin" had been used showed a marked reduction in the respiratory minute volume. They showed more or less marked cyanosis, and therefore were suffering from anoxæmia.

To secure surgical anæsthesia the concentration of ether vapour had to be increased; so much so that the vapour now became a respiratory irritant. After any abdominal operation the patient had a long period in which the respiratory centre remained depressed and the respiratory exchange was considerably reduced.

Referring to the theory that pulmonary collapse was caused by the presence of a mucous plug in a bronchus, Dr. Darling said that certain difficulties arose in that connexion. Lee, Ravdin, Tucker and Pendergrass had taken a mucous plug from a patient with apneumatosis and introduced it into the bronchus of a dog; if they abolished the cough reflex pulmonary collapse occurred. When the plug was removed the lung did not necessarily aerate again. This fact was difficult of explanation. Further, if collapse was due to a mucous plug, why was pulmonary collapse so frequently unilateral? Dr. Darling said that he had met with very few cases in which the condition was bilateral, and could recall only one other case in which the bases of both lungs had been collapsed.

Dr. Darling thought that massive collapse of the lung was very rare; he had met with only about five cases himself. Partial collapse was extremely frequent but would not be found unless it was looked for.

DR. A. DISTIN MORGAN also thanked those who had spoken. He said that he had recently been in America and had spent some time with two men who had done much original work on the subject of massive collapse of the lung—Waters and Rovestine. These two men had probably used more cyclopropane than anyone else in the world. They had found that in the presence of high percentages of oxygen absorption of gases from the occluded lung was five-sixths complete in two minutes. When the oxygen concentration was high, therefore, there was a tendency to massive collapse of the lung, irrespective of what the anæsthetic might be. Another factor was the actual plugging; this had been recognized by Dr. Bye. Then again there was the lowered respiratory exchange, caused by several factors; one was premedication, which raised the threshold for stimulation of respiration by carbon dioxide. After operations performed with closed circuit anæsthesia, when the carbon dioxide was washed out of the lungs the respiratory exchange was very low for a time. Therefore, when a high concentration of oxygen was withdrawn the patient was unable to get sufficient out of the air by breathing and became anoxic. Dr. Morgan said that although he did not think that massive collapse of the lung occurred more frequently in America, it was more frequently observed there; anæsthetists were looking for it and their treatment was very prompt. Everyone knew that in urgent and extensive massive collapse the patient could die in a few moments. There was a great need for oxygen and patients died from anoxia. They should be given high concentrations of oxygen, which would keep them alive. The next step in treatment was the induction of extensive hyperpnœa by the exhibition of a 5% to 7% concentration of carbon dioxide. The respiratory exchange was increased very many times. Hyperpnœa was induced with the object of removing the plug; or in the absence of a plug the hyperpnœa, by increasing the respiratory exchange, increased aeration. If these measures did not produce results rapidly bronchoscopy had to be used at once. Patients in imminent danger of death were often relieved by these three methods. In less severe cases the first two methods of treatment were used. Such cases were quite frequent, and Dr. Morgan had seen a number. The diagnostic points were: first, symptoms of cyanosis and an increase in the rate of respiration, often associated with a rise in temperature; secondly, a difference in the movements of the two sides of the chest. The physical signs heard by the stethoscope were often misleading. Radiological examination was helpful when it revealed narrowing of the costo-phrenic angle and displacement of the heart. In conclusion Dr. Morgan said that he agreed with the statement of both speakers, that if medical men were on the look-out for massive collapse of the lung they would find it more frequently. Many of the post-operative complications classed as pneumonia actually began as collapse of the lung.

DR. R. S. STEEL said that he wished to refer to one point that had been mentioned by Dr. Bye, and that was the occurrence of atelectasis in asthmatics. It was often overlooked. Dr. Steel thought that so-called pneumonia of short duration was actually massive collapse of the lung. The essential lesion in allergy was a localized œdema, and that occurred in the bronchi. Dr. Steel pointed out that that was believed to be one cause of massive collapse. In the asthmatic this essential lesion was present or constantly in a state of approximation; massive collapse was likely to occur, and did occur. Dr. Steel said that about two months earlier a patient had walked into his surgery with signs of consolidation in the base of the right lung. He looked quite well and his temperature was normal; the physical signs in the whole of the consolidated area were indicative of pneumonia.

The patient was an asthmatic whom Dr. Steel had been treating for some time. He had a slight cough, which had increased a little. Dr. Steel had told the patient that if he were seen by another practitioner he would probably be put into hospital. The patient was seen three days later by a suburban practitioner, who sent him to hospital with a diagnosis of pneumonia. The "pneumonia" was better in a few days. Dr. Steel further quoted the case of a boy whom he had examined about eighteen months earlier. At that time the boy had proudly said that he had had pneumonia seventeen times. He was obviously telling an unintentional lie; he really had had recurrent attacks of massive collapse of the lung.

Dr. Bye, in reply to Professor Lambie, said that he had read a little about the allergic theory, but partly because of the time allotted to him, and more particularly because his courage had failed, he had not alluded to it. Dr. Bye thanked Dr. Steel for his remarks, which would partly answer Professor Lambie's question. Bergamini and Shephard had said that their *post mortem* findings in the lung had shown a condition closely resembling angio-neurotic œdema. With regard to the physical signs of massive collapse of the lung, Dr. Bye could not answer Professor Lambie to Professor Lambie's satisfaction. It had been suggested that in the earlier stages of collapse the breath sounds would be likely to be absent; at a later stage, as some air entered the lung, tubular breath sounds would probably be heard. A change in the position of the secretion might have something to do with the varied nature of the physical signs. Dr. Bye then said that what he had read had led him to believe that there was a considerable amount of truth in what Dr. Darling had said; though massive collapse of the lung was not common, partial or incomplete collapse was of frequent occurrence. Jackson had pointed out that incomplete collapse might be of just as much significance in leading up to post-operative pneumonic complications. In conclusion Dr. Bye said that he was glad to hear that Dr. Walker also had difficulty at times in determining the position of the heart. Further in reply to Dr. Walker he said that he could not explain why in some cases the onset of massive collapse of the lung might be delayed until as long as one week after the operation.

Dr. Marshall, in reply, expressed his gratitude to those who had joined in the discussion, especially those who had confirmed some of his assertions. Dr. Marshall thanked Professor Lambie for what he had said concerning the part played by the anæsthetic in the production of massive collapse of the lung. Professor Lambie had pointed out that the anæsthetic should be blamed only when blockage occurred during or soon after operation, not when it occurred a week later. Dr. Marshall was in agreement with what Grey Turner had said concerning the undesirability of taking a patient to the operating theatre in a comatose condition and allowing him to remain so for hours after his return to the ward; at the same time Grey Turner had said that coughing and vomiting after operation were good for the patient and helped him to empty his lungs. Dr. Marshall regarded that statement as a confession of defeat and an exhibition of sadism. Dr. Morgan had referred to the work of Waters, of Madison. Dr. Marshall said that Waters was a recognized authority and his work was often quoted. Dr. Marshall, in reference to Dr. Morgan's remarks concerning the washing out of the lungs with carbon dioxide during closed circuit anæsthesia, said that the inevitable "dead space" in all gas anæsthetic apparatus was a safeguard against carbon dioxide depletion.

A MEETING of the Queensland Branch of the British Medical Association was held at the Mater Misericordiæ Children's Hospital on February 3, 1939. The meeting took the form of a series of clinical demonstrations by members of the honorary staff.

Giant-Cell Tumour.

DR. G. W. MASON showed a boy, aged eight years, who was suffering from a giant-cell tumour of the left humerus. This was a typical example of a lesion, often seen in children, which might give rise to a great deal of doubt as to what procedure was best for its treatment. The case was interesting, as the radiographs showed the progress over a number of years, with the results following the surgical treatment and later irradiation. The series illustrated the migration of the lesion along the shaft and explained how the latent bone cyst, which was sometimes found towards the centre of the shaft of a long bone in an older patient, originated.

In 1935, when five years old, the boy was brought in complaining of pain and swelling in the upper part of the left arm following a twist. An X ray examination disclosed a fusiform expansion of thin cortical bone in the metaphyseal region, trabeculæ extending across the cyst, and a pathological fracture. Slightly increased density at the margin showed an attempt at spontaneous healing. Records showed that twelve months previously a similar accident had occurred. Dr. Mason said that such a lesion in a young child immediately below the epiphyseal line of the upper end of the humerus, femur or tibia (the major long bones) was a bone cyst. Occasionally the bone cyst was of a more acute variety; it became larger and spread, and was often mistaken for a giant-cell tumour. Pathological fracture was usually the most frequent reason for the patient's consulting the physician. A tendency to unite might occur, and the arrest of the lesion without obliteration of the cavity would result in the latent bone cyst referred to above.

In this boy the lesion had progressed, and in March, 1935, an operation—curettage and swabbing out of the cyst wall with carbolic acid—was performed. The biopsy report was "giant-cell tumour of the humerus". The result six weeks later was excellent; there was good regeneration and the cavity was filling with new calcified tissue. Twelve months later evidence of fresh activity was found and X ray treatment was given. The lesion extended ten centimetres along the humerus; the arm was only five centimetres wide. Moderate irradiation was used in April, 1936, and July, 1936. In March, 1937, a further course of X ray treatment was indicated. The technique used was a weekly dose with potential of 150 kilovolts, 0·5 millimetre of copper filter, anterior and posterior fields, four doses of 250 *r* each. Progress was shown in X ray films taken subsequently.

At the time of the meeting the boy was aged eight years. The epiphysis had laid down new bone and the cystic area was near the middle of the shaft. The epiphyseal area had been protected during X radiation and there was no difference in the length of the left humerus from that of the right. A small cystic area could be seen at the distal part. It was doubtful if there was any activity there; there was no expansion and no cortical thinning of bone was evident, but the area would be given a further repetition of treatment to encourage healing.

Slides of similar cases were shown. The first was of a bone cyst with a pathological fracture at the upper end of the femur, in a lad, aged sixteen years. A splint was applied, the leg was kept at rest, and healing progressed. The cyst had arisen in juxtaposition to the epiphyseal line. The next film was that of a pathological fracture in a femur, due to a bone cyst in a girl of eighteen years. A *coxa vara* deformity had developed, due to the adjustment of the weakened shaft to stresses.

A typical bone cyst, almost identical with the earliest X ray pictures of the boy shown at the meeting, was shown in the next film, the lesion occupying the shaft of the humerus just below the unossified epiphyseal line. The film showed destruction of the medullary bone, expansion of the cortex, and pathological fracture. The last-mentioned condition, Dr. Mason said, was the first clinical sign in 45% of cases.

The next slide illustrated a giant-cell variant of a bone cyst on the shaft side of the epiphyseal line at the lower end of the radius. Its condition before and after radiation treatment was shown. The true giant-cell tumour was shown in the next slide. Here the lesion had arisen

after the union of the epiphysis, and the asymmetry and extent of invasion of the whole of the end of the shaft, including the epiphyseal portion, were shown. The last figure showed the malignant changes which might occur in giant-cell tumours. It showed the spread across the tibio-fibular attachment into the fibula with a fungating mass through the sinus in the tibia.

Dr. Mason said that the differential diagnosis included five lesions: (i) Rarely a gumma of the bone might simulate a cyst, but the periosteal involvement and the reaction to the Wassermann test assisted in the making of a diagnosis. (ii) Brodie's abscess was a smaller lesion, rarely expanded the bone, and was accompanied by signs of infection. (iii) Giant-cell tumour was the most likely mistake. It was a lesion of adults, involved the epiphysis instead of the metaphysis and had a greater tendency to progress. (iv) Chondromyxoma was the most difficult to distinguish from this more benign lesion. It produced an area of bone destruction very similar to a cyst, and when occurring as a central lesion in a long bone, a very rare finding, the chondroma was more finely loculated. Bending deformity, indicating a chronic disturbance to which the weakened bone had become gradually adjusted, favoured a benign bone cyst. Adjacent areas of increased density, suggesting a healing reaction, also favoured a bone cyst. (v) Fibrocystic disease showed more diffuse involvement; hypercalcæmia and a lowered phosphorus value were present in these cases.

Dr. Mason showed a schematic drawing taken from *The Journal of Bone and Joint Surgery*, Number 4, October, 1938, indicating the location in respect to the epiphyseal line of a simple bone cyst, a giant-cell variant of a bone cyst, and a giant-cell tumour.

DR. A. V. MEEHAN said that the condition had been known as giant-cell sarcoma and there had been many unnecessary amputations. There were no metastases from it. Dr. Meehan had seen cases in which irradiation alone gave excellent results; he had also used curettage in cases in which the disease had recurred. The actual curetting should be done meticulously under a very good light; Dr. Meehan used pure carbolic acid followed by absolute alcohol to swab out the bone and had no recurrences with this. After the curettage a dose of irradiation was given and all the patients had done very well. He allowed one patient to go for years under the impression that the condition never caused metastases, but now he told the patient to have the irradiation, as there was a slight possibility of metastasis.

DR. GEORGE TAYLOR said that the condition was supposed to be absolutely benign, but it might become malignant in the mandible. Metastases never occurred.

Apophysitis.

DR. J. R. S. LAHZ showed a boy, aged fourteen years, who for two years had been limping on the left leg. He complained of slight pain in the heel on walking. There was great atrophy of the left lower limb when compared with the right; no paresis or joint defects were seen. There was broadening of the heel, which was tender on the medial side of the epiphysis of the *os calcis*; both feet showed mild *pes cavus*. X ray examination showed thickening and sclerosis of both calcaneal epiphyses, the bone of which was very dense on each side. The diagnosis was of apophysitis of the *os calcis* on each side, with symptoms only on the left side. The interest of the case was in the pronounced limp and the great wasting of the left limb in a comparatively mild lesion and the absence of symptoms on the other side.

DR. A. V. MEEHAN said that in his experience a similar condition could be present, without symptoms, in the other foot. The length of the rest period depended on the symptoms, and in some cases at least two months' rest was necessary, in plaster; then the patient might be given a walking plaster. Occasionally patients were affected in a similar way in later life, the shape of the modern heel causing pain from pressure. For treatment, sometimes it was sufficient to remove the stiffening from the heel; in other cases operation was necessary. The sharp upper and outer borders of the *os calcis* were removed and there would be no further trouble.

Syndactylism.

DR. A. V. MEEHAN showed a boy, aged seven years, who had complete webbing of the fingers and thumb. A series of plastic operations had been done on the hand. Dr. Meehan pointed out the importance of cutting a basal flap which covered the position of the web with sound skin, as the essential point in any operations for webbed fingers. Unless this flap was sound the webbing gradually reformed from the normal end forward. In its present condition thumb and fingers were free and the intrinsic muscles were being repaired.

Anterior Poliomyelitis.

Dr. Meehan then showed three patients suffering from anterior poliomyelitis.

The first was a girl, four years of age, who was admitted to hospital on July 12, 1938. Twelve weeks previously she had complained of pain in both legs and was disinclined to walk. Soon after that she was admitted to the Rockhampton Hospital and was discharged a fortnight before admission to the Mater Misericordiæ Children's Hospital. She had been well since discharge, but her gait had been affected. On examination she was found to have a limp with the weight on the left leg. The right leg was slightly abducted, lifted high, and "flung" at the knee muscles. On July 31, 1938, the child was having reeducation; there was weakness of the left quadriceps and hamstrings and also of the extensors of the toes of both feet. On August 15, 1938, the condition was improving, and on September 11, 1938, it was satisfactory. On January 20, 1939, the child was allowed to walk with aid, and on January 31 walked alone; the muscle tone was improving.

Dr. Meehan's second patient was a girl, aged one year and four months, who was admitted to hospital on January 18, 1939. She gave a history of having been well until four weeks previously; she had begun to walk when one year old. Four weeks previously it had been suddenly noticed that the child could not bear any weight on the right leg, and on being stood up she would immediately fall. Since then the condition had remained the same, though she made some attempt to pull herself up. She could not walk and dragged the right leg when crawling. There had been a slight fretfulness noticed about ten days before the onset of symptoms, but otherwise nothing abnormal. On examination it was found that the right leg was not moved freely and that the foot was plantar flexed. There was little power of resistance in the quadriceps or hamstrings; dorsiflexion of the foot was impossible, and there was contraction of the *tendo Achillis*. On January 26, 1939, a scrim cast was made; by January 31 there was some relaxation of the *tendo Achillis*, but the condition was unchanged otherwise. A plaster was applied to the legs.

Dr. Meehan's third patient was aged three years, and was admitted to hospital on December 31, 1939. On December 7 the patient, a boy, had begun to vomit. Next day he began to complain of pain in the back of the neck and later in the back. He was drowsy, but no paresis was noted. He was taken to hospital on December 10, and the mother thought that the paresis occurred about a week after admission. He was discharged from hospital on February 3, 1939. On examination he seemed to lack movement of the left arm and right leg. On January 5, 1939, a scrim cast was prepared. The arms were put on right-angled splints. On January 31 there was considerable improvement and the back was less stiff. The left leg was still weak, but he could lift it up. The *tendo Achillis* was relaxing; the right leg was very poor. He appeared to have full power in the muscles controlling the right hand, thumb and fingers; there was slight power in the elbow flexors. There appeared to be some flexion power in the fingers of the left hand, but practically no power in the left arm. In the left leg all the muscles below the knee

were well; there was some power in the left quadriceps, but he could not hold the limb against gravity. The hamstrings were working. There was some power in the abductors and rotators of the thigh and *gluteus maximus*. In the right leg there was no power in the quadriceps or hamstrings. There was no power in the gluteus or *tensor fasciæ latæ*. The abdominal muscles showed some power. There was severe spinal rigidity.

Pyloric Stenosis.

DR. P. A. EARNSHAW showed a patient who was suffering from pyloric stenosis. The patient, a boy, was the seventh in the family and was quite well till six weeks of age, when he had projectile vomiting. The baby was constipated and passed little urine. Visible gastric peristalsis was pronounced, but despite several careful examinations no pyloric tumour could be felt. A barium meal was given. X ray pictures revealed very little barium as having passed through the pylorus in four hours.

The baby was then given a freshly prepared aqueous solution (1 in 10,000) of "Eumydrin". The vomiting immediately lessened and the baby became comfortable. During the first day following the use of "Eumydrin" the baby vomited once, and twice the next day. The child did not vomit on the third day, but vomited once on the fourth. The next and last vomiting occurred on the twelfth day. The child since then had behaved as a normal baby.

Dr. Earnshaw stated that this was the fifth child he had treated with "Eumydrin", and all had done well. They were fortunate in Australia in that the great majority of cases of pyloric stenosis seen were recognized early. When there was little dehydration there was no reason why these children should not be treated in their own homes. A nurse was not necessary in such cases. When, however, dehydration was pronounced, admission to hospital would undoubtedly be necessary. The treatment was usually continued for one or two months.

Subarachnoid Hæmorrhage.

Dr. Earnshaw's next patient was a boy who was sent into hospital as possibly suffering from pyloric stenosis. The child had been well till he was six weeks old, when he suddenly developed occasional projectile vomiting. Definite gastric peristalsis had been observed prior to his admission to hospital. He entered hospital when seven weeks old. While he was in hospital gastric peristalsis was not observed, nor was a pyloric tumour felt. However, in spite of the vomiting, his fontanelle was noticed to be under increased tension and rather spongy.

A lumbar puncture was carried out, the cerebro-spinal fluid containing old blood. The child apparently had suffered from a subarachnoid hæmorrhage at birth. Through blockage of the Pacchionian bodies and arachnoid villi by the red blood cells the cerebro-spinal fluid tension was raised. The increased intracranial tension no doubt was the cause of the gastric symptoms. A second lumbar puncture was done a few days later and the baby made a complete recovery.

Epituberculosis.

Dr. Earnshaw finally showed a patient who was possibly suffering from epituberculosis. The patient was a girl, aged three and a half years. The mother stated that the child had had a slight cough for some time. The day before her admission to hospital she coughed a great deal. The night before admission she coughed up blood on two occasions, and again on the morning of her admission to hospital.

When she was admitted to hospital it was noticed that she was pale, with a harsh cough, but the breathing was not distressed. Coarse râles were heard in both bases, especially the left. She was given an anæsthetic with a view to the making of a bronchoscopic examination, but a severe hæmorrhage occurred during the induction of anæsthesia and before any examination was attempted. The child became collapsed and pulseless. The blood was aspirated. Artificial respiration was successful. The patient was then transferred to the medical ward. Four days later a bronchoscopic examination was made, but no abnormality was detected.

Dulness over the upper portion of the left lung could then be demonstrated by percussion. Breath sounds were somewhat impaired, but there were no adventitious sounds.

During the first week after the patient's admission to hospital the temperature varied between 98° and 101° F. Thereafter there were occasional rises of temperature to 99° F. There was a constant slight cough and the child did not look well, though she did not appear to be very ill. Tubercle bacilli could not be detected in a smear from the pharynx or fæces. The Mantoux test elicited a positive reaction in a 1 in 100 dilution, but no reaction in 1 in 1,000 and 1 in 10,000 dilutions. The Casoni test elicited no reaction. An X ray picture showed an area of opacity in the left apical region in the neighbourhood of the mediastinum.

Dr. Earnshaw thought that epituberculosis should be considered in the diagnosis of this condition. The history of a young ailing, though not seriously ill, child with an unproductive cough, slight fever, the failure to find tubercle bacilli in the sputum or fæces, a positive result to a Mantoux test (though not strongly positive in this case), the impairment of the percussion note over the upper lobe, and the radiological photograph provided a typical picture. Against the diagnosis of epituberculosis were the attacks of hæmoptysis, which Dr. Earnshaw had not seen mentioned in the reports that he had read on this condition.

The X ray picture showed a homogeneous shadow extending from the left of the hilus to the parietal pleura. The upper border of the shadow reached to the apex, while the lower border had a clear-cut margin with a slight concavity downwards. This margin was considered by many to coincide with the interlobar septum.

Dr. Earnshaw said that there had been several interpretations of such X ray appearances in epituberculosis. One was that they were due to collapse of a lobe or part of a lobe, the collapse resulting from pressure on a bronchus by a tuberculous gland. Another suggestion was that the phenomenon was an allergic one and that there was a non-specific infiltration of the lung in the vicinity of a tuberculous gland. Some people regarded the condition as a tuberculous infiltration either by spread from the gland or by ulceration into the bronchus. Other ideas were that the condition was due to a mediastinal effusion or unresolved pneumonia. The importance of this condition of epituberculosis lay in the subsequent course of the disease. The signs might remain for weeks or months without any deterioration in health and then gradually disappear, this recovery being a constant feature.

Post-Graduate Work.

LECTURES ON CIVILIAN WAR CASUALTIES.

THE Melbourne Permanent Post-Graduate Committee has arranged a course of lectures dealing with the care of civilian war casualties as follows:

Monday, July 24, at 4.15 p.m.[1] "Pathological Demonstration of Features of Bomb and High Explosive Wounds, including Bacteriology", Professor Peter MacCallum, "Surgical Aspects", Mr. C. Gordon Shaw.

Thursday, July 27, at 4.15 p.m.—"General Principles of Treatment of High Explosive and Bomb Injuries, Shock, Hæmorrhage, Resuscitation", Mr. Victor Hurley. "Blood Transfusion under War-Time Conditions", Dr. Ian Wood.

Monday, July 31, at 4.15 p.m.—"Gas: History of its Use in War; the Evolution of the Respirator", Emeritus Professor W. A. Osborne.

[1] The first lecture was given on Thursday, July 20, by Dr. H. N. Featonby and Dr. J. Newman Morris.

Thursday, August 3, at 4.15 p.m.—"The Treatment of Gas Casualties", Dr. Charles Adey.

Friday, August 4, at 8.30 p.m., at the United Service Institution, Victoria Barracks (entrance in Coventry Street; telephone number, only Central 10170).—Practical demonstration of gas mask and the gas chamber.

Monday, August 7, at 4.15 p.m.—"The Crushed Limb, Fractures and Joint Injuries", Dr. W. G. D. Upjohn. "Preparation for, Care in and Methods of Transport", Dr. Eric Cooper.

Wednesday, August 9, at 4.15 p.m.—"Thoracic Injuries", Dr. W. A. Hailes. "Abdominal Wounds", Mr. Balcombe Quick.

Friday, August 11, at 4.15 p.m.—"Head Wounds", Sir Alan Newton. "Injuries to Face and Neck", Mr. Fay Maclure.

All lectures, except the practical demonstration at the United Service Institution, will be held at the Medical Society Hall, 426, Albert Street, East Melbourne.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 37, of June 15, 1939.

AUSTRALIAN MILITARY FORCES.

Second Military District.

Australian Army Medical Corps.

To be Major—Captain K. S. Richardson, 5th May, 1939. *To be Captain (provisionally)*—Arthur Alexander Moon, 21st April, 1939. Captain (provisionally) R. H. Macdonald is brought on the authorized establishment, 1st February, 1939. The provisional appointments of Captains J. G. Woods and M. R. Joseph are confirmed.

Australian Army Medical Corps Reserve.

To be Honorary Captains—Ernest McAustin Steel, 3rd April, 1939, and Nathan John Clements, 12th April, 1939. *To be Honorary Lieutenant*—2nd Lieutenant R. B. Parker, 22nd May, 1939.

Third Military District.

Australian Army Medical Corps.

Captain S. Crawcour is appointed from the Reserve of Officers (A.A.M.C.) supernumerary to the establishment pending absorption, 8th May, 1939. *To be Captains (provisionally) supernumerary to the establishment pending absorption*—Thomas Edward Lowe, John MacDonald Agar, Arthur David Mawson and Brian Lister Hellings, 8th May, 1939. Captains C. D. Donald, J. S. T. Stevens and C. G. McAdam and Captains (provisionally) R. S. Smiebert, W. D. Refshauge, D. J. Shale, A. C. Mendelsohn, H. I. Gibb, M. M. Rosefield, J. S. Peters, G. J. M. Stoney, C. H. Johnston and M. A. Rees are brought on the authorized establishment, 1st May, 1939.

Australian Army Medical Corps Reserve.

To be Honorary Major—John Bell Ferguson, 2nd May, 1939. *To be Honorary Captains*—Alfred Ernest Brauer, Francis Edmund Browne, Roderick Euan George MacLean, David Alan Kidd, Ronald William Douglas Fisher, Francis Syndal Coombs, Theophil Johannes Friedrich Frank, William Leslie Colquhoun, Vernon Brentani Brenton, Howard Lyell Andrews, Frank Robison Kerr, Frank David Burke, George Bankin Bearham, George Frederick Bennett, Solomon Rose, Harold Charles Ralph Carter, Cecil Nathaniel Love Cantor, John Berchmans Devine, Arthur Roberts Moreton, William Raymond Dudley Griffiths, Thomas Glass Millar, Francis Gerald Donovan, David George Alsop, Edward Eric Bottomley, Leslie Algernon Ivan Maxwell, Frank Ward Farmer, Alexander Edward Lincoln, Sidney Kadish Crownson, Charles James Officer Brown, Noel Tracey Bull, Norman Hudson Luth, Ian Donald McInnes, Ernest Danby, Hugh Henry Martin, William Harold James Moore, Ernest Kirsner, John Joseph Laurence McDonald, Tom Allistair Falconer Heale, George Murch Haydon, Ewen Thomas Taylor Downie, Norman Anderson Longden, Edgar Samuel John King, Douglas Ian Hart, George Robin Adlington Syme and Leonard Hunt Ball, 15th May, 1939.

Fourth Military District.

Australian Army Medical Corps.

Major A. L. Dawkins, Captain R. N. Reilly and Captains (provisionally) A. H. White, E. P. Cherry, J. A. Game, R. C. Angove, W. M. Irwin, M. C. Newland, R. G. Champion de Crespigny, D. W. Brummitt and G. W. Verco are brought on the authorized establishment, 28th April, 1939.

Australian Army Medical Corps Reserve.

To be Honorary Captain—Robert Kevin Wilson, 4th May, 1939.

Sixth Military District.

Australian Army Medical Corps.

Honorary Captain N. B. G. Abbott is appointed from the Australian Army Medical Corps Reserve and to be Captain, 22nd December, 1938. (This cancels the notification respecting the appointment of this officer which appeared in Executive Minute No. 74/1939, promulgated in *Commonwealth Gazette* No. 13, of 16th March, 1939.) Captain K. J. Fagan is transferred to the Reserve of Officers (A.A.M.C.), 16th March, 1939.

Australian Army Medical Corps Reserve.

To be Honorary Major—Ralph Whishaw, 13th May, 1939. *To be Honorary Captain*—Arthur Edwin Ernest Grounds, 1st May, 1939.

Obituary.

HENRY HAVELOCK ELLIS.

WE regret to announce the death of Dr. Henry Havelock Ellis, which occurred on July 10, 1939, at London, England.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Eglitzky, Ben-Zion Charles, M.B., 1939 (Univ. Sydney), Balmain and District Hospital, Balmain.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Gilbert, Philip, M.B., B.S., 1935 (Univ. Sydney), "Apsley", Princes Avenue, Vaucluse.

Joseph, Neil, M.B., B.S., 1937 (Univ. Sydney), 43, Kambala Road, Bellevue Hill.

Mackenzie, John Kenneth, M.B., B.S., 1939 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Mankin, Winifred Roby, M.B., B.S., 1939 (Univ. Sydney), Royal Alexandra Hospital for Children, Camperdown.

O'Halloran, Max Anthony, M.B., B.S., 1938 (Univ. Sydney), c.o. Dr. V. L. Bourke, Merewether, Newcastle.

Sewell, Arthur Kenneth, M.B., B.S., 1939 (Univ. Sydney), 5, Deakin Avenue, Haberfield.

Speight, Richard John Joseph, M.B., 1931 (Univ. Sydney), 37, Johnston Street, Annandale.

Reid, William Lister, M.B., B.S., 1931 (Univ. Adelaide), 54, Cranbrook Road, Bellevue Hill.

The undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Strauss, Maurice, M.D.S., 1936 (Padua), 223, Elgin Street, Carlton.

The undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Cuming, Ian Harry, M.B., B.S., 1938 (Univ. Melbourne), Alfred Hospital, Prahran.

Lane, William Reade, M.B., B.S., 1938 (Univ. Melbourne), Royal Melbourne Hospital, Melbourne, C.1.

The undermentioned has applied for election as a member of the South Australian Branch of the British Medical Association:

Shepherd, David Wickham, M.B., B.S., 1938 (Univ. Adelaide), Adelaide Hospital, Adelaide.

The undermentioned have been elected members of the South Australian Branch of the British Medical Association:

Bishopverder, Ernest, German Licence, Berlin, 1928, M.B., L.R.C.P. and S. (Edinburgh), L.R.F.P.S. (Glasgow), 1938, Hawker.

Plummer, Rex Grose, M.B., B.S., 1937 (Univ. Adelaide), Strathalbyn. (In the issue of July 1, 1939, at page 50, this name was wrongly given as Plummer, Rex Grosse.)

Books Received.

ILLUSTRATIONS OF REGIONAL ANATOMY, by E. B. Jamieson, M.D.; Second Edition; 1939. Section VI: Upper Limb, containing 42 plates; Section VII: Lower Limb, containing 52 plates. Foolscap 4to. Price: Section VI, 7s. 6d. net; Section VII, 10s. net.

GYNÆCOLOGY, by H. H. Schlink, M.B., Ch.M., F.R.A.C.S.; 1939. Australia: Angus and Robertson Limited. Medium 8vo, pp. 572, with illustrations. Price: 32s. 6d. net.

ESSENTIALS OF FEVERS, by G. E. Breen, M.D., Ch.B., D.P.H., D.O.M.S. (R.C.P. London, R.C.S. England); 1939. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 286, with illustrations. Price: 7s. 6d. net.

PHYSIOTHERAPY IN MEDICAL PRACTICE, by H. Morris, M.D., D.M.R.E.; 1939. London: Edward Arnold and Company. Demy 8vo, pp. 283, with illustrations. Price: 12s. 6d. net.

Diary for the Month.

JULY 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JULY 26.—Victorian Branch, B.M.A.: Council.
JULY 27.—New South Wales Branch, B.M.A.: Branch.
JULY 27.—South Australian Branch, B.M.A.: Branch.
JULY 28.—Queensland Branch, B.M.A.: Council.
AUG. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 2.—Victorian Branch, B.M.A.: Branch.
AUG. 2.—Western Australian Branch, B.M.A.: Council.
AUG. 3.—South Australian Branch, B.M.A.: Council.
AUG. 4.—Queensland Branch, B.M.A.: Branch.
AUG. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 11.—Queensland Branch, B.M.A.: Council.
AUG. 15.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 16.—Western Australian Branch, B.M.A.: Branch.
AUG. 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi-xvii.

BRITISH MEDICAL AGENCY OF QUEENSLAND PROPRIETARY LIMITED: Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	Wiluna Hospital. All Contract Practice Appointments in Western Australia.

Editorial Notices.

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